#19345

Sam.l Lewis M.D.

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# TRANSACTIONS

OF THE

# PATHOLOGICAL SOCIETY

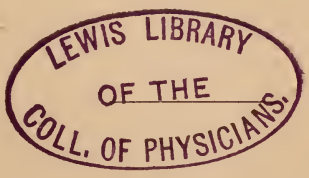
## OF PHILADELPHIA.

VOLUME TENTH.

CONTAINING THE REPORT OF THE PROCEEDINGS FROM SEPTEMBER, 1879, TO JULY, 1881.

EDITED BY

CHAS. B. NANCREDE, M.D.,
SURGEON TO THE P. EPISCOPAL HOSPITAL AND TO ST. CHRISTOPHER'S HOSPITAL FOR CHILDREN; RECORDER OF THE SOCIETY.




PHILADELPHIA:
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The present volume includes the Transactions of the Society from September, 1879, to June, 1881, inclusive.

The papers by Dr. Nancrede, on The Pathology of Fracture of the Cranial Base, etc. (p. 2); of Dr. F. P. Henry, On Some of the Causes Determining the Nature of Pleural Effusion, etc. (p. 209); of Dr. J. M. Da Costa, On Some Points in the Pathology of Tubercle (p. 229); and of Dr. H. F. Formad, On the Etiology of Tumors (p. 248), were read by the respective authors at the semi-annual conversational meetings of the Society, they having been appointed to open the proceedings on those evenings.

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# Former Presidents.

SAMUEL D. GROSS, M.D., LL.D., D.C.L. Oxon., LL.D. Cantab., elected 1857.
RENÉ LA ROCHE, M.D., elected 1858.
ALFRED STILLÉ, M.D., elected 1859, '61, and '62.
EDWARD HARTSHORNE, M.D., elected 1860 and '63.
J. M. DA COSTA, M.D., elected 1864, '65, and '66.
JOHN H. PACKARD, M.D., elected 1867 and '68.
S. WEIR MITCHELL, M.D., elected 1869.
JOHN ASHHURST, JR., M.D., elected 1870.
JAMES H. HUTCHINSON, M.D., elected 1871 and '72.
WILLIAM PEPPER, M.D., elected 1873.
H. LENOX HODGE, M.D., elected 1876.
S. W. GROSS, M.D., elected 1879.

# OFFICERS AND COMMITTEES

OF THE

# Pathological Society of Philadelphia.

---

**President.**

S. W. GROSS, M.D.

ELECTED OCTOBER, 1879, FOR THREE YEARS.

ELECTED AT THE ANNUAL MEETING, OCTOBER, 1881.

**Vice-Presidents.**

JAMES TYSON, M.D.,
J. S. COHEN, M.D.,
F. P. HENRY, M.D.,
J. E. MEARS, M.D.

**Secretary.**

S. F. HAZLEHURST, M.D.

**Recorder.**

C. B. NANCREDE, M.D.

**Treasurer.**

M. S. FRENCH, M.D.

**Curator.**

CARL SEILER, M.D.

**Committee on Publication.**

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J. H. BRINTON, M.D., J. H. HUTCHINSON, M.D.,
C. B. NANCREDE, M.D. (ex-officio).

**Committee on Morbid Growths.**

C. SEILER, M.D., M. LONGSTRETH, M D.,
J. H. C. SIMES, M.D., E. O. SKAKESPEARE, M.D.,
H. F. FORMAD, M.D., C. B. NANCREDE, M.D.,
J. H. MUSSER, M.D.

**Business Committee.**

M. LONGSTRETH, M.D., (Sec.), E. O. SHAKESPEARE, M.D.,
J. TYSON, M.D., C. B. NANCREDE, M.D.,
W. H. PARRISH, M.D.

# LIST OF MEMBERS.

Members marked O. M. are of those originating the Society.
Members marked N. R. are Non-resident.

*Elected*

1857 AGNEW, D. HAYES, Professor of Surgery in the University of Pennsylvania, 1611 Chestnut Street. (O. M.)

1871 ALISON, ROBERT H., 250 South Seventeenth Street, one of the Physicians to the Hospital for Nervous Diseases.

1864 ALLEN, HARRISON, resigned December 11th, 1879.

1869 ALLIS, OSCAR H., resigned February 24th, 1881.

1865 ANDREWS, THOMAS H., 1117 Spruce Street.

1871 ASHBRIDGE, WILLIAM, resigned December 11th, 1879.

1867 ASHHURST, FRANCIS, Mount Holly, N. J. (N. R.)

1861 ASHHURST, JOHN, JR., Professor of Clinical Surgery in the University of Pennsylvania, 2000 West De Lancey Place.

1863 ASHHURST, SAMUEL, 2308 De Lancey Place.

1871 ATLEE, WASHINGTON L., Jr., forfeited membership by non-payment of dues, December 10th, 1874.

1866 BACHE, T. HEWSON, 233 South Thirteenth Street.

1878 BAER, B. F., 218 South Twentieth Street.

1876 BARR, J. W., forfeited membership by non-payment of dues, December 8th, 1881.

1880 BARTHOLOW, ROBERTS, Professor of Materia Medica in the Jefferson Medical College, 1509 Walnut Street.

1867 BARTLES, WILLIAM H., Insane Department Pennsylvania Hospital, West Philadelphia. (N. R.)

1869 BARTON, J. M., 1344 Spruce Street.

1873 BEECHER, A. C. W., 1222 Walnut Street.

1870 BELL, J. R. F., forfeited membership by non-payment of dues, December 10th, 1874.

1871 BENNETT, WM. H., 332 South Fifteenth Street.

1863 BENTON, C. H. (N. R.)

1875 BERNARDY, EUGENE P., resigned September 22d, 1881.

1870 BERTOLET, ROBERT MORRIS. Died, 1882.

1871 BETTS, THOMAS, Branchtown, Pa. (N. R.)

*Elected*

1860 Bishop, C. S., died.

1866 Black, J. J., New Castle, Delaware. (N. R.)

1863 Boardman, C. H., St. Paul, Minnesota. (N. R.)

1880 Bœnning, Henry C., 1208 Race Street.

1857 Boker, Charles S., one of the Surgeons to St. Joseph's Hospital, 1622 Chestnut Street. (O. M.)

1868 Bolles, Lucius S., died August 15th, 1873.

1877 Bradford, T. Hewson, resigned December 23d, 1880.

1873 Bray, Daniel, forfeited membership by non-payment of dues, December 8th, 1881.

1857 Brinton, John H., Professor of the Practice of Surgery and Clinical Surgery in the Jefferson Medical College, Philadelphia, 1423 Spruce Street. (O. M.)

1874 Brubaker, A. P., 217 North Thirteenth Street.

1874 Bruen, Edward T., one of the Visiting Physicians to the Philadelphia Hospital, 1531 Chestnut Street.

1871 Buck, William Penn, resigned October 23d, 1873.

1867 Burnett, C. H., resigned December 8th, 1881.

1872 Cathcart, James H., died March 5th, 1874.

1879 Cathcart, T. H., died March 28th, 1882.

1871 Cheston, C. Morris, West River, Maryland. (N. R.)

1865 Cheston, D. Murray, one of the Physicians to the Children's Hospital, 1835 Chestnut Street.

1871 Clark, Leonardo S., 1505 Girard Avenue.

1866 Cleemann, Richard A., one of the Physicians to St. Mary's Hospital, 340 South Twenty-first Street.

1872 Cohen, J. Solis, Lecturer on Laryngoscopy and Diseases of the Throat and Chest in the Jefferson Medical College, 1431 Walnut Street.

1877 Collins, James, 538 Marshall Street.

1869 Corbit, William B., Washington, D. C. (N. R.)

1877 Cruice, R. B., 731 North Seventeenth Street.

1871 Curtin, Roland G., Lecturer on Physical Diagnosis in the University of Pennsylvania, 332 South Seventeenth Street.

1857 Da Costa, J. M., Professor of Theory and Practice of Medicine in the Jefferson Medical College, 1700 Walnut Street. (O. M.)

1859 Darby, John T., New York City. (N. R.)

1876 Darlington, W. L., 146 North Twentieth Street.

1857 Darrach, James, 5094 Green Street, Germantown. (O. M.)

1868 Darrach, William, died January 28th, 1881.

1877 Deaver, R. W., 5075 Main Street, above High, Germantown.

1877 Drysdale, T. M., northeast corner Sixteenth and Arch Streets.

*Elected*

1866 Duer, Edward L., one of the Obstetricians to the Philadelphia Hospital, 1704 Arch Street.

1870 Duhring, L. A., Clinical Professor of Dermatology in the University of Pennsylvania, 1411 Spruce Street.

1876 Dulles, C. W., 114 South Fortieth Street.

1860 Dunton, W. R., 5059 Germantown Avenue, Germantown.

1863 Dyer, Ezra, Pittsburg, Pennsylvania. (N. R.)

1875 Engel, Hugo, forfeited membership by non-payment of dues, February 23d, 1882.

1876 Eskridge, J. T., 1614 North Sixteenth Street.

1881 Fenton, T. H., 1335 Arch Street.

1857 Fischer, Emil, southeast corner Sixth and Brown Streets. (O. M.)

1863 Fish, Augustine H., died August 3d, 1872.

1876 Fisher, Henry M., 919 Walnut Street.

1857 Forbes, William S., one of the Surgeons to the Episcopal Hospital, 1405 Locust Street. (O. M.)

1869 Ford, William H., 1622 Summer Street.

1878 Formad, H. F., 105 South Thirty-fourth Street.

1871 Fox, Charles, resigned December 10th, 1874.

1880 French, M. S., 1423 Walnut Street.

1868 Garretson, James E., 1537 Chestnut Street.

1871 Gerhard, George S., Ardmore. (N. R.)

1870 Getchell, Frank H., Clinical Lecturer on Diseases of Women in the Jefferson Medical College, 1432 Spruce Street.

1877 Gilliard, Louis, died November, 1878.

1877 Goldsborough, C. B. (N. R.)

1866 Goodell, William, Clinical Professor of Diseases of Women in the University of Pennsylvania, Preston Retreat, Twentieth and Hamilton Streets.

1868 Goodman, H. Ernest, resigned February 23d, 1882.

1868 Graham, James, resigned January 9th, 1879.

1867 Grier, Matthew J., 1531 Spruce Street.

1868 Gross, Ferdinand H., one of the Surgeons to the German Hospital, 931 North Broad Street.

1857 Gross, Samuel D., Emeritus Professor of Surgery in the Jefferson Medical College, southeast corner Eleventh and Walnut Streets. (O. M.)

1858 Gross, Samuel W., Professor of the Principles of Surgery and Clinical Surgery in the Jefferson Medical College, 1112 Walnut Street.

1869 Grove, John H., one of the Surgeons to St. Mary's Hospital, 1330 Arch Street.

1872 Gruel, Theodore H. E., 612 Spruce Street.

1874 Guitéras, John. (N. R.)

*Elected*

1871 Hale, George J., forfeited membership by non-payment of dues, March 23d, 1876.

1857 Hall, A. Douglass, resigned October 9th, 1873. (O. M.)

1875 Hand, Frank C., died September 1st, 1881.

1866 Hare, Horace Binney, died March 21st, 1879.

1868 Hargadine, R. W. (N. R.)

1859 Harlan, George C., resigned January 12th, 1882.

1874 Harris, Charles M., 1715 Locust Street.

1858 Harris, Robert P., resigned January 9th, 1873.

1857 Hartshorne, Edward, 331 South Broad Street. (O. M.)

1859 Hartshorne, Henry, resigned October 12th, 1864.

1867 Hassler, Ferdinand A., resigned February 9th, 1871.

1869 Hatfield, Nathan L., one of the Visiting Surgeons to the Philadelphia Hospital, 501 Franklin Street.

1868 Hays, I. Minis, resigned January 13th, 1881.

1879 Hazlehurst, S. F. (N. R.)

1873 Hearn, Joseph, one of the Surgeons to the Philadelphia Hospital, 324 Catharine Street.

1870 Henry, Frederick P., one of the Physicians to the Episcopal Hospital, 721 Pine Street.

1866 Herbert, Theodore. (N. R.)

1879 Hess, Robert, resigned January 12th, 1882.

1857 Hewson, Addinell (O. M.), forfeited membership by non-payment of dues, December 22d, 1881.

1880 Hewson, A., Jr., 1421 Locust Street.

1874 Hickman, N. B., forfeited membership by non-payment of dues, December 8th, 1881.

1858 Hodge, H. Lenox, died June 10th, 1881.

1871 Hoffman, Washington Atlee, died September 20th, 1874.

1860 Hopkins, Henry St. George. (N. R.)

1876 Hopkins, W. B., resigned October 14th, 1880.

1877 Horwitz, Theodore, died December 12th, 1877.

1870 Houston, James P. S., Savannah, Georgia. (N. R.)

1866 Howe, Herbert M., resigned February 28th, 1878.

1859 Hoyt, William D. (N. R.)

1879 Huidekoper, R. S., resigned October 28th, 1880.

1857 Humphries, George H., New York City. (O. M., N. R.)

1868 Hunter, Charles T., resigned October 14th, 1880.

1870 Hutchins, E. R. (N. R.)

1858 Hutchinson, James H., one of the Physicians to the Pennsylvania Hospital, 2019 Walnut Street.

1866 Ingham, James V., resigned December 11th, 1879.

1876 Ingram, T. D., forfeited membership by non-payment of dues, December 8th, 1881.

*Elected*

1874 Jamison, Edward, St. Louis, Mo. (N. R.)
1866 Jenks, William F., died October 31st, 1881.
1875 Johnson, Russell H., resigned December 8th, 1881.

1859 Kane, John K., Wilmington, Delaware. (N. R.)
1874 Keating, John M., resigned December 22d, 1881.
1857 Keating, William V. (O. M.), forfeited membership by non-payment of dues, September 14th, 1864.
1857 Kealer, William, resigned January 28th, 1875. (O. M.)
1866 Keen, William W., Professor of Artistic Anatomy, Pennsylvania Academy of Fine Arts, 1729 Chestnut Street.
1875 Kelly, Joseph V., 4257 Main Street, Manayunk.
1858 King, William, died.
1879 Kirkbride, J. J., 35 South Nineteenth Street.
1869 Knight, Samuel R., resigned October 28th, 1880.

1867 Lambdin, Alfred C., forfeited membership by non-payment of dues, December 10th, 1874.
1871 Landis, Henry G., Niles, Ohio. (N. R.)
1872 La Roche, C. Percy, Nice, France. (N. R.)
1857 La Roche, René. (O. M.) Died December 9th, 1872.
1870 Leach, Alonzo L., forfeited membership by non-payment of dues, December 22d, 1881.
1869 Leaman, Henry, 1031 Vine Street.
1860 Lee, Charles C., New York City. (N. R.)
1861 Leedom, John M., Pulaski Avenue, Germantown.
1857 Levick, James J., resigned October 13th, 1861. (O. M.)
1868 Lewis, Francis W., 2016 Spruce Street.
1869 Lewis, Fred. W., died 1873.
1875 Lewis, Morris J., one of the Physicians to the Episcopal Hospital, 1216 Walnut Street.
1857 Lewis, Samuel, Aldine Hotel. (O. M.)
1877 Little, William S., 219 South Seventeenth Street.
1859 Livezy, Edward, forfeited membership by non-payment of dues, September 14th, 1864.
1876 Loder, P. E., forfeited membership by non-payment of dues, February 9th, 1882.
1873 Longenecker, Jerome, forfeited membership by non-payment of dues, 1875.
1870 Longstreth, Morris, one of the Physicians to the Pennsylvania Hospital, 1416 Spruce Street.
1870 Loughlin, J. Eneu, forfeited membership by non-payment of dues, May 9th, 1878.

1874 Mann, Charles H. (N. R.)
1867 Markoe, James, 2217 St. James Place.

*Elected*

1868 Martin, George. (N. R.)
1874 Matteson, Charles C., died.
1865 Maury, Frank F., died 1879.
1870 Maxwell, J. Gordon, forfeited membership by non-payment of dues, December 10th, 1874.
1866 McArthur, John A., forfeited membership by non-payment of dues, December 8th, 1881.
1861 McCall, C. A., resigned.
1880 McClellan, G., 1352 Spruce Street.
1868 McClure, W. Wallace, resigned September 10th, 1878.
1873 McCoy, A. W., Pittsburg, Pa. (N. R.)
1864 Mears, J. Ewing, one of the Surgeons to St. Mary's Hospital, 1429 Walnut Street.
1873 Meigs, Arthur V., one of the Physicians to the Pennsylvania Hospital, 1322 Walnut Street.
1857 Meigs, John Forsyth, 1208 Walnut Street. (O. M.)
1874 Miller, C. K. I., resigned December 28th, 1876, died 1878.
1878 Mills, C. K., 1502 Columbia Avenue.
1857 Mitchell, S. Weir, one of the Physicians to the Hospital for Nervous Diseases, 1523 Walnut Street. (O. M.)
1876 Montgomery, E. C., southeast corner Twentieth and Jefferson Streets.
1857 Morehouse, George R., 227 South Ninth Street.
1857 Morton, Thomas George (O. M.) forfeited membership by non-payment of dues, September 14th, 1864.
1859 Moss, William, Chestnut Avenue, Chestnut Hill.
1871 Muhlenberg, Frank, 1911 Chestnut Street.
1869 Müller, August F., 4544 Germantown Avenue.
1880 Musser, John H., Chief of Medical Dispensary, University Hospital, 37 Powelton Avenue.
1873 Musser, Milton B., Fortieth and Locust Streets.
1869 Mustin, J. Burton, died 1871.

1869 Nancrede, Charles B., one of the Surgeons to the Episcopal Hospital, Surgeon to St. Christopher's Hospital for Children, 2109 Pine Street.
1879 Neff, J. S., 214 South Fifteenth Street.
1872 Newsham, Stanley P., 1640 South Fifth Street.
1868 Norris, Herbert, resigned January 8th, 1880.
1868 Norris, Isaac, resigned November 12th, 1874.
1869 Norris, J. C., resigned February 10th, 1876.
1861 Norris, William F., Clinical Professor of Ophthalmology in the University of Pennsylvania, 1526 Locust Street.

*Elected*

1875 O'Hara, Michael, 31 South Sixteenth Street.
1874 Osgood, Hamilton. (N. R.)

1857 Packard, John H., one of the Surgeons to the Episcopal Hospital, 1928 Spruce Street. (O. M.)
1868 Pancoast, William H., Professor of Anatomy in the Jefferson Medical College, 1100 Walnut Street.
1873 Parish, William H., Professor of Anatomy in the Woman's Medical College, 324 South Seventeenth Street.
1875 Parks, Edward L. (N. R.)
1867 Parry, John S., died 1876.
1870 Paul, Comegys, Resident Physician Eastern Penitentiary.
1875 Paul, James, resigned January 22, 1880.
1857 Penrose, R. A. F. (O. M,), forfeited membership by non-payment of dues, September 14th, 1864.
1865 Pepper, George, died September 14th, 1872.
1865 Pepper, William, Professor of Clinical Medicine in the University of Pennsylvania, 1811 Spruce Street.
1879 Perkins, F. M., 1428 Pine Street.
1870 Porter, William G., Jr., one of the Surgeons to the Presbyterian Hospital, 1223 Spruce Street.

1878 Raub, J. A., 927 North Tenth Street.
1874 Reed, Henry B., resigned December 9th, 1880.
1859 Reed, Thomas B., one of the Surgeons to the Presbyterian Hospital, 1427 Walnut Street.
1871 Rex, George A., 2118 Pine Street.
1869 Rex, Oliver P., forfeited membership by non-payment of dues, December 10th, 1874.
1877 Reynolds, A. S., forfeited membership by non-payment of dues, February 9th, 1882.
1864 Rhoads, Edward, died January 16th, 1871.
1878 Rich, T. C., resigned January 13th, 1881.
1869 Richardson, Elliott, resigned December 8th, 1881.
1863 Richardson, Joseph G., one of the Visiting Physicians to the Presbyterian Hospital, 3238 Chestnut Street.
1857 Richardson, T. G., New Orleans, La. (O. M., N. R.)
1876 Risley, S. D., Lecturer on Ophthalmoscopy in the University of Pennsylvania, 1630 Walnut Street.
1868 Ritz, Charles M. (N. R.)
1880 Roberts, A. S , 131 South Fifteenth Street.
1876 Roberts, J. B., 1118 Arch Street.
1876 Roland, O. (N. R.)
1874 Ronaldson, William D., 4017 Locust Street.

*Elected*

1877 Rudolph, B. J., forfeited membership by non-payment of dues, February 23d, 1882.

1867 Santee, Eugene J., 532 North Sixth Street.

1863 Savery, William, resigned February 11th, 1869.

1869 Schell, H. S., resigned December 8th, 1881.

1874 Seiler, Carl, 1608 Pine Street.

1870 Seyfert, Theodore H., resigned December 27th, 1877.

1870 Shaffner, Charles, 23 South Sixteenth Street.

1876 Shakespeare, E. O., Lecturer on Refraction and Accommodation and Operative Ophthalmic Surgery in the University of Pennsylvania, 1336 Spruce Street.

1869 Shapleigh, Elisha B., 658 North Eighth Street.

1868 Sherwood, Thomas H., forfeited membership by non-payment of dues, December 10th, 1874.

1878 Shoemaker, J. V., 1031 Walnut Street.

1871 Simes, J. H. C., Lecturer on Histology in the University of Pennsylvania, 2033 Chestnut Street.

1868 Sinkler, Wharton, one of the Physicians to the Hospital for Nervous Diseases, 1534 Pine Street.

1881 Skillern, P. G., 429 South Broad Street.

1881 Skillern, S. R., 3416 Baring Street.

1858 Smith, A. H., Lecturer on Obstetrics at the Philadelphia Lying-in Charity, 1419 Walnut Street.

1863 Smith, E. A., Aldine Hotel.

1859 Smith, F. G., resigned November 13th, 1873. Died April 6th, 1878.

1876 Smith, R. M., resigned December 9th, 1880.

1875 Smith, Stanley. (N. R.)

1876 Smythe, F. G., died 1879.

1871 Sparks, George W., forfeited membership by non-payment of dues, December 10th, 1874.

1866 Spooner, Edward A., forfeited membership by non-payment of dues, December 10th, 1874.

1871 Starr, Louis, resigned October 14th, 1880.

1857 Stillé, Alfred, Professor of Theory and Practice of Medicine in the University of Pennsylvania, 3900 Spruce Street. (O. M.)

1870 Strawbridge, George, resigned June 10th, 1875.

1869 Stryker, Samuel S., one of the Obstetricians to the Philadelphia Hospital, 3713 Walnut Street.

1879 Taylor, J. M., northwest corner Twenty-second Street and St. Alban's Place.

1866 Thomas, Charles H., 1807 Chestnut Street.

1868 Thomson, William, one of the Surgeons to Wills Ophthalmic Hospital, 1502 Locust Street.

*Elected*

1870 Tinkham, J. H., United States Navy. (N. R.)
1866 Townsend, George D. (N. R.)
1868 Townsend, R. M., died.
1864 Turner, A. P. (N. R.)
1863 Tutt, Charles Pendleton, died May 11th, 1866.
1863 Tyson, James, Professor of General Pathology and Morbid Anatomy in the University of Pennsylvania, 1506 Spruce Street.

1871 Valdivieso, R. (N. R.)
1870 Van Harlingen, Arthur, resigned December 8th, 1881.
1879 Van Valzah, W. W., 1704 Arch Street.

1874 Warder, Wm. H., one of the Obstetricians to the Philadelphia Hospital, 810 North Broad Street.
1867 Watson, E. W., 146 North Twentieth Street.
1866 Weightman, John F., resigned April 27th, 1871.
1876 Weir, A. H., resigned April 14th, 1881.
1875 West, Franklin, died.
1875 Whelen, Alfred, resigned December 23d, 1880.
1873 White, J. William, one of the Visiting Surgeons to the Philadelphia Hospital, 222 South Sixteenth Street.
1868 Willard, De Forrest, Lecturer on Orthopædic Surgery in the University of Pennsylvania, 1818 Chestnut Street.
1865 Williams, Horace, resigned December 11th, 1879.
1875 Williamson, Jesse, resigned October 13th, 1881.
1867 Wilson, Ellwood, resigned.
1880 Wilson, H. A., 331 South Twelfth Street.
1869 Wilson, James C., one of the Visiting Physicians to the Philadelphia Hospital, 1437 Walnut Street.
1867 Wilson, James F., 1010 Race Street.
1869 Wilson, J. H. (N. R.)
1873 Winslow, W. H., resigned December 28th, 1876.
1864 Wistar, Thomas, resigned February 13th, 1867.
1875 Wolford, W. S., 133 South Thirteenth Street.
1863 Wood, Horatio C., Jr., forfeited membership by non-payment of dues, March 23d, 1876.
1865 Woods, D. Flavel, one of the Physicians to the Presbyterian Hospital, 151 North Fifteenth Street.
1857 Woodward, J. J., U. S. A., Washington, D. C. (O. M., N. R.)
1872 Worthington, David J., forfeited membership by non-payment of dues, February 9th, 1882.

1876 Zeigler, G. W. (N. R.)

# CORRESPONDING MEMBERS

1866 Bumstead, Freeman J. Died 1879.

1859 Clark, Alonzo, Professor of Theory and Practice of Medicine in the College of Physicians and Surgeons, New York.

1859 Dalton, J. C., Professor of Physiology in the College of Physicians and Surgeons, New York.

1860 Ellis, Calvin, Professor of Theory and Practice of Medicine in the Medical Department of Harvard University, Boston, Massachusetts.

1858 Flint, Austin, Sr., Professor of Theory and Practice of Medicine in Bellevue Hospital Medical College, New York.

1858 Hammond, William A., Member of the National Academy of Sciences, New York.

1860 Isaacs, Charles E., Brooklyn. Died June 16th, 1860, æt. 48.

1878 Jackson, J. B. S., Boston. Died 1879.

1860 Reeves, James E., Wheeling, West Virginia.

1861 Rokitansky, Carl. Died 1878.

1860 Watson, John, New York. Died June 3d, 1869, æt. 56.

1859 Worthington, Wilmer, West Chester, Pennsylvania. Died 1873.

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# REPORT.

## I. THE OSSEOUS SYSTEM.

### 1. *Cystic giant-celled sarcoma of the head of the tibia.*

Presented by Dr. Samuel W. Gross.

A single woman, æt. 20 years, presented herself at the out-patients' department of the Jefferson Medical College Hospital on the 24th of January, 1880, on account of a huge tumor of the upper epiphysis of the left tibia, of two years' duration. About three years previously she fell and struck her knee upon the cobble-stones; after which she experienced a sense of weakness in the joint, but did not discover a swelling until after the lapse of twelve months, when she observed a tumor, of the volume of a hen's egg, just below the lower border of the patella. For eighteen months its growth was so slow that its size had only doubled itself; but six months ago she again fell upon her knee, after which the tumor increased rapidly, and the subcutaneous veins began to enlarge. At times, when the weather was damp, she experienced sharp, shooting, or stinging pains, but, as a rule, she was free from suffering. There was some heat in the swelling; the skin was not changed in texture or color; the veins were as large as the little finger; there was some tenderness on manipulation; the consistence of the mass was firm and elastic, except at its outer side, where there was marked parchment-like crepitation. The leg could be flexed upon the thigh, but the power of extension was slight.

After amputation through the lower third of the thigh, by Dr. Levis, at the Pennsylvania Hospital, the head and upper portion of the shaft of the tibia were found to have been converted into a periosteo-osseous capsule, which was occupied by a firm mass, the centre of which was of a yellowish-white tint, while the peripheral portion, for an average width of an inch and a half, was of a dull-red color and pervaded by cysts, some of which were an inch and a quarter in diameter, and con-

tained a citron-colored fluid. The tumor measured six inches and three-quarters in length by five inches in width, and was limited above by the articular cartilage, which was sound.

Minute examination of sections taken from the red portion of the mass disclosed an exquisite example of giant-celled sarcoma, and the commencing cystic changes could be readily followed through mucoid transformation of the multinucleated elements. The central portion of the growth had undergone advanced fatty metamorphosis.

*March 25th*, 1880.

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## 2. *The etiology of fractures of the cranial base, and the anatomico-pathological reasons for their fatality.*

Read by Dr. C. B. Nancrede.

Gentlemen: When asked to prepare a paper for this evening, I felt how utterly inadequate was the time which remained before the 23d of September in which to write one worthy your consideration. As one of the authors of the amendment to the Constitution whereby the conversational meetings were inaugurated, I felt that it would ill befit me to refuse to fill the gap left by the declination of one so much better fitted for the task as my friend Dr. J. Ashhurst, Jr.

Upon due reflection, the subject to which I propose to call your attention this evening has seemed eminently fitting for the purpose. Although, of course, to some the views I intend advancing are neither altogether new nor original, yet, to judge by the literature of the subject, few practically realize their importance.

Let it be clearly understood that the major part, if not all, of what I propose saying is not original; but, owing to the seeming ignorance of most writers upon the subject, the old views need restating. The ordinary view held, and, judging from the results, taught, is that these injuries are either grave from the amount of violence causing them, or from the important vital structures contiguous to the injured bones, which are liable to similar traumatic lesions. Now, I would state, at the outset, that I consider this view to be very incorrect, and that could we change the structure of the skull so as to render its *base* like its *vault*, extensive shatterings could not occur; or, if they did, no more evil results would follow than in an ordinary fracture of the vault. When my explanations are given, this apparently bold statement will

appear, I trust, justified; although, strictly speaking, it is only approximately correct. It is, however, sufficiently accurate for the sake of impressing what I believe to be an important fact.

Of course it will be understood that I do not deny that death is the direct result of basal injuries when they are such complete crushes as to divide the brain-case into several distinct segments, with extensive lacerations of the brain-substance. Perhaps the clearest way to explain my views will be to take a case such as I have seen myself, or such as Hilton relates, as a text.

First, however, let us classify the various basal fractures, and then define clearly which are dealt with in this paper. First, then, we have fractures of the base resulting from direct violence, such as a thrust from a cane or umbrella in the orbit. A violent blow on the nose, which fractures its bones, may also break the cribriform plate of the ethmoid, as in these specimens which I show you. Then, too, the condyles of the lower jaw have been driven through the glenoid fossæ of the temporal bones by direct violence applied to the former. Besides such instances of direct violence, there are others where I maintain that it is in reality the fracturing force. I suppose that many will take exception to this statement; still, it is true, and is constantly taught, although not formulated as I have done. Suppose a man falls from a great height upon his feet, what results? Why, practically, the condyles of his occipital bone are struck violently through the medium of the spinal column,—more violently, indeed, than otherwise could occur, because *plus* all the amount of the vibrating structures of the body, except such as are diverted or checked by the various tutamina, struck just as *directly* for mechanical purposes as if with a sledge-hammer.

Again, if a man falls from a height and strikes full upon the vault in some comparatively yielding substance, as soft earth, the weight of the body again strikes, through the medium of the spine, a violent blow upon the occipital condyles, thus fracturing them. This statement of such injuries being due to direct force may appear clearer if I say that a blow is merely the exciter of vibrations, and that we must consider the point whence they start as in reality the *point of impact.*

Then, again, a third class present themselves, where the fissure starts in the vault and merely reaches the base by direct continuity. The head may be also caught between two opposing forces, when a general crush involving the base will ensue. Finally, we have those said to be produced by *contre-coup.* If we mean by this a fracture

upon the side opposite to the point struck, unless this point be about the centre of the vault, this certainly is unlikely. For instance, a violent blow upon the right parietal boss will not produce a fracture at the left parietal eminence, but at the base of the cranium, through one or both petrous bones. I do not deny that a blow in this situation may produce laceration of the brain-substance exactly opposite to the point struck, because the brain is practically homogeneous, and vibrations *cæteris paribus* are conveyed through it equally, while the skull *does not* convey vibrations equally well in all directions. Again, the statement that the vibrations which are excited in the vault travel by the shortest route to the base is absolutely incorrect. They reach the base by the "shortest anatomical route," indeed, but that is very different from the shortest route, and is often quite a long one. Again, the statement made by a recent author that a blow upon the occiput may break the petrous portion of the temporal bones, because the "basilar process rests against the apices of these petrous bones," is a mistake, as the force which breaks these bones does not reach them through the basilar process, but the vibrations pursue another course, as will be presently explained.

The mistake here made is in failing to remember that the various portions of the skull are of varying thicknesses, and are so constructed for specific ends. If the vault were of even thickness and density, as well as all of the base except its central portion, and if the skull were also a true sphere, *then* true fracture by *contre-coup* of the base would obtain, for the vibrations would travel from the point struck equally in all directions until, meeting at the weak central basal portion, it would be disrupted. But the skull is of unequal thickness, it is not a sphere, and certain portions of its base, although frequently broken, are in reality the densest and strongest.

It is unquestionably true that the portion of the skull struck determines which of the fossæ of the cranial base the fracture will traverse. This I hope to explain by certain anatomical peculiarities of the skull-structure. This fact relative to the point struck determining the site of the fracture, has been proved by the experiments of Aran and the investigations of Prescott Hewett.

Some writers have been so hard pushed to explain fractures at a distance from the part struck, that they have actually ascribed them to the vibrations communicated to the *brain*, which then acts as the fracturing force. Thus, Prof. Longmore considers that certain fractures of the orbital plates of the frontal bone, caused by gun-shot wounds of

distant parts of the skull, are actually produced by the vibrations of the brain. In this view Otis concurs. Dr. Harrison Allen, in an article on fractures of the cranium, in Hays's *American Journal of the Medical Sciences* for January, 1874, gives an explanation of such cases, instancing that of President Lincoln. I regret to feel compelled to dissent from his conclusions. In the proper place the case will be related, with Dr. Allen's comments upon it, and my own reasons for dissent.

Before proceeding to the anatomical demonstrations which I wish to make, let me refresh your memory by a homely illustration as to the direct proportion of the force of vibrations to the amount of the bony or other tissue struck. If one end of a wire of fine calibre be held in the hand, and the other, after being laid upon an anvil, be struck never so hard, no appreciable vibrations will be perceived; yet they are present. Let the same experiment be tried with a crowbar, and I think few of us would care to suffer the unpleasant jar that would result. Now, just this same thing occurs in the skull: where thin the vibrations are imperceptible, where thick they are marked. But the base of the skull, having to support the superincumbent weight of the skull and brain, and afford a firm basis for the movements of the lower jaw and of the head on the spine, as well as to protect the important nervous structures connected with it, must be massive, at least in parts. These advantages during health have compensating disadvantages when man is subjected to violence: indeed, in his ordinary movements this massiveness of portions of his cranial base would be very disadvantageous were it not for certain wise provisions presently to be spoken of. It is hardly necessary for me to refer to the ordinary tutamina of the brain, such as the hair, scalp, pericranium, and dura mater, acting, as the latter two do, as a wash-leather lining and cover would to a bell. It will, however, be essential to speak of certain processes of the dura mater.

I will now give briefly a history of a case of fracture of the base, which I propose to use as my text, in conjunction with the general relation of similar cases given by Mr. Hilton in his remarkable lectures upon the cranium. My friend Dr. Charles Wirgman called upon me one evening some months back, requesting me to see in consultation with him a young boy of some nine to ten years, who, he thought, was suffering from a fracture of the skull. I am indebted to Dr. Wirgman for the following outlines of the case. Four days previously he had been desired to see the patient, who had walked home, assisted by his

playmates, after having fallen on his head while playing leap-frog. When Dr. Wirgman saw him he was suffering from slight symptoms of concussion, the exact details of which are of no moment. The following day he seemed better, only complaining of some headache. Upon the third day the improvement was *very* marked, nothing remaining but some headache. He was very desirous of being allowed to sit at the window, and I know that the doctor's injunction as to perfect quietude was not complied with. The patient, when called upon to evacuate his bowels, went out as a matter of course to the yard, and walked about the apartment when so inclined. Only a few hours after Dr. Wirgman's last visit he was hastily summoned: but before going he called upon me to request my advice and assistance. The patient's residence was at a short distance, but when we arrived he had been dead some little time. He had been—as far as could be ascertained—attacked with vomiting, which was followed by eclampsia, rapidly terminating life. I expressed the opinion that the patient had had a fracture of the base of the cranium, and that perfect quietude might possibly have averted the fatal results. By the courtesy of Dr. Wirgman and Dr. Lee, the then coroner's surgeon, I was present at the autopsy. An insignificant ecchymosis was detected over the right mastoid region, which, it is probable, resulted from infiltration of blood from the fracture. Upon reflecting the skin, what at first sight looked merely like the normal masto-occipital suture, with a little coagulated blood occupying its depression, was seen. Upon a closer scrutiny this proved to be a fracture separating the synarthrosis. The test upon which I relied was the impossibility of removing the line of coagulated blood effused between the fragments, while the same collected in a suture can always be wiped or washed away. Upon removing the calvarium, a line of fracture was discovered which traversed the right petrous bone, extending posteriorly from the occipito-mastoid suture forward. A small blood-clot of a few lines in extent lay near the fracture, but there was nothing that could be called meningitis: indeed, the brain and its membranes were unusually healthy-looking.

Hilton makes the following pertinent remarks, which I transcribe verbatim:—

"I have known it to happen that a person, having been exposed to external violence, which has led to a fracture of the base of the skull, and feeling pretty well a few days after the accident, has expressed a desire to get up and leave his sick-chamber, which his medical attendant has been indiscreet enough to allow him to do, or which he has

done of his own accord, without the knowledge or consent of his medical adviser. After moving and walking about, however, for a short time, he has soon complained of headache, has been attacked with sickness and vomiting, afterwards has had confusion of his ideas, and, finally, has fallen into a state of unconsciousness, in which, after three or four days, he has expired."

The first point of mechanical interest in the structure of the brain-case is its ovoid form, to which is ascribed a large proportion of the security of the brain. To a considerable extent this is true; but it is a matter of profound surprise to me how one important point is apparently completely overlooked. Take up any work upon anatomy, and the author will doubtless expatiate upon the strength secured by the peculiar form of the brain-case, any segment being a section of a spheroid. If an injury—as a fracture—were only produced by a *crushing* force, this would be an incontrovertible proposition; if, upon the other hand, a large part of the damage is done by setting up *vibrations* in the bones themselves, then the form of the skull only conduces in a slight degree to its immunity from fracture. An arch that will sustain uninjured hundreds of tons' *pressure*, if vibrations are set up in it, may be readily *fissured* in many directions, although its *form* may remain unchanged. The arched form of the skull, then, merely resists the tendency of the bones to be pressed inward when a blow is struck; after that the fracturing force expends itself in producing vibrations, which cause the fracture. Any brittle body capable of fracture will be so injured whenever the *vibrations become violent enough to overcome the cohesion between the particles composing that body.*

Let us first examine the anatomy of the child's skull, as it may give us some hints which will prove useful. I suppose that I may safely say that the base of a *young* child's skull has never been fractured by anything but direct force, or that more than *one* bone of the vault has ever been broken unless the force was actually applied to it. Now, why is this so? Examine an infant's skull, and you will see that each bone is isolated from every other by membrane,—than which there is no better arrester of vibrations. Again, as you will readily see by a careful examination of any young parietal or frontal bone, but specially of one say at the sixteenth week *in utero*, the most projecting portions (those, of course, which are most apt to strike the ground first in a fall), are the thickest, while extending from these centres are radii of bone becoming more and more delicate. Each of the fine terminal points of these radii is enveloped by the membrane, which, by its ossi-

fication, forms the bone. Recall now my homely illustration of the vibrations of the wire and the crowbar. Do you not see that the vibrations start in the denser bone, but, being conducted on, are propagated by thinner and thinner bone (*i. e.*, a structure less and less capable of vibrating strongly) until they are finally safely conducted off to the terminal radii of the bone, which are *completely enveloped in membrane*, and are thus totally arrested? By this you can readily see that the injury inflicted upon one bone of an infant's head by a blow never passes beyond the one bone struck.

These efficient means for the arrest of vibrations explain the immunity from injury enjoyed by infants and children, where, considering the strength of their skulls, the injuries they receive with impunity are otherwise inexplicable. This is, indeed, fortunate, when we consider how "mobile" is the nervous system of the infant, and how seriously injuries of it are resented.

What, therefore, should we learn from this? That one of the most efficient means for arresting vibration in bone is to have it enveloped closely by a membranous tissue.

Turning to the adult skull, I would call your attention to the following points. The projecting parietal bosses, with the frontal eminences, are not so prominent, and, instead of being the thickest portions of the vault, they are often the thinnest. The eight component bones of the brain-case are more or less co-ossified, and are in bony contact. Let us remove the calvarium of both the infant and the adult. In the infant the various bony projections are certainly present, but the brain looks as if it would fit fairly. The base of the adult's skull looks as if nothing could be better fitted to lacerate the delicate brain, and is not at all correspondent in form. Here in front are the projecting anterior clinoids, there behind the posterior projections of the same name; here is a deep depression, there an elevation. If a cast of the base of the brain were taken, it would plainly show that there exists but little correspondence between this portion of the case and its contents. A blow on this practically solid ovoid of bone formed by the skull will infallibly set up vibrations, which will pass in any and every direction. Here is an organ of delicate structure which apparently has to lie upon a rugged and uneven bed. The ordinary motions of walking, much more those of running or jumping, would apparently imperil the integrity of the vital nerve-centres. What, then, upon the whole renders the brain the best-protected organ in the body? Again, let me say

that the hair, scalp, etc., are excluded from consideration as tutamina in our present remarks.

The mechanical effect of the diploë, however, must not be ignored, especially in view of some statements with regard to it made by Dr. Allen in the paper already cited. In the first place, he denied the correctness of the usual descriptions of there being two tables of compact tissue in the skull. On the contrary, he maintains that there is but one, viz., the internal, and that the so-called external table is only modified cancellous tissue.

I cannot agree with him, for, call it what you may, it is still a much more compact, dense structure than the subjacent diploë, and as such a vastly better conductor of vibrations. On the other hand, I entirely agree with him in thinking that the inner is the best and principal conductor of vibrations. Again, Dr. Allen cites the easily demonstrable fact that there is an actual absence of the diploë in certain portions of several of the cranial bones. The sites specially to be noted where this important element is absent are the cerebellar fossæ, the roof of the glenoid fossa, the greater part of the squamous plate of the temporal bone, the orbital plates of the frontal, and the floor of the lateral sinuses, as they approach the base of the brain. Here also my views clash with those of the author cited. He says that in these situations the internal and external tables are in contact, or, more correctly, that *both* diploë *and* external table are absent. The importance of this will appear when he goes on to say that a blow struck upon any portion of the skull, if not of sufficient violence to pass the damper of the diploë to the internal table, is conducted to more distant points along the diploë itself, not by the external table, which he maintains does not exist. For me, however, it does exist, and is of great importance, for the following reasons. By Dr. Allen's explanation, both in injury and in the ordinary normal movements, the slighter vibrations are confined to the diploë, while the more violent simply do damage by affecting the vitreous table despite the diploë and plus whatever vibrations may be present in the latter. I, however, would liken the two tables to two insulated wires, the insulating medium being the diploë. As long as the diploë is present, either of the two tables may vibrate separately, or both in differing degrees. At those places, however, where the diploë is absent, the two conductors of vibrations are in contact and the circuit is completed, the spark leaps across, and we have a shock; *i. e.*, to return from illustration to fact, the vibrations

of differing intensity meet, and a fracture takes place, subject to certain modifying influences next to be referred to.

To return to our question, What renders the brain the best-protected organ of the body? This was when speaking of the apparent want of adaptation between the base of the brain and of the brain-case. The answer is, these very irregularities and projections, which seem fitted only to lacerate the delicate structures lying superimposed. Just where the greatest dissimilarity exists between the brain and its case, viz., in the central portion, in the normal state, the brain is distinctly lifted off from the bones by a stratum of fluid,—the cerebro-spinal fluid,—which serves, as Mr. Hilton points out, as a "water-bed." By means of this the cerebellum does not come into contact even with the posterior cerebral fossæ, and the central portions of the cerebrum are held off at a distinct interval from the bones. Some such provision is absolutely necessary for the ordinary normal movements to proceed in safety. Without something similar, even walking—much more running and jumping—would inevitably result in fatal laceration of the brain-pulp. But its usefulness does not end here. What better means of arresting vibrations originating in the bony parietes which might otherwise prove prejudicial to the brain? Surely, nothing.[1]

Let me pass around this specimen of the base of the skull with its calvarium. By holding it up to the light you will see that there are certain thin portions mapped out by certain thicker ridges. This will be clear from the different transmission of the rays of light. These thicker portions, in general terms, converge towards the petrous portions of the temporal bone, the anterior and posterior clinoid processes, and the crista galli of the ethmoid. Examine now with me the anatomical peculiarities of the parts mentioned. The anterior clinoids are surrounded by dura mater, and are immersed in the cerebro-spinal fluid. The same description will answer for the posterior clinoids. The apex of the petrous portion of the temporal bones, and a portion of their inner margins, seem, the former in contact with the sphenoidal body, the latter with the basilar process of the occipital bone. In reality, the apex of the petrous bone is separated from the body of the sphenoid by the plate of cartilaginous tissue filling the foramen lacerum medius, while the inferior petrosal sinus lies in a groove formed partly by the petrous and partly by the basilar process of the occipital bone.

[1] Of course the vibrations which meet at the base of the skull may neutralize one another.

Finally, the crista galli is the point of attachment of the falx cerebri, in the commencement of which it is imbedded. Let the skull now be set in vibration by a blow, and, instead of reaching the base by the "nearest" route, the vibrations will follow the nearest one, or *all*, of these *anatomical* routes.

I think, then, that I have demonstrated that these apparently useless and dangerous projections have a useful purpose, for each serves as the terminal discharge-point for the accumulated vibrations which *must* follow, by virtue of mechanical laws, the best *conductors*, viz., the densest portions of the bone,[1] which have been all stated to converge to the points mentioned. These points are provided with such efficient dampers of vibrations that all the ordinary and many of the extraordinary vibrations are rendered innocuous. Indeed, had this properly come within the scope of my paper, I think I could demonstrate that the ordinary vibrations are positively put to a physiological use.

Let me now apply the knowledge gained from these anatomical studies to the question of fracture of the cranial base.

The statement, then, made by Aran and others—that fracture of the base is invariably accompanied by a fissure of the vault, which in reality is the starting-point of the fracture—is, I think, incorrect, from the foregoing facts as to the conduction of vibrations, even if we exclude these specimens and the case I have related. If Aran's view were correct, the greatest force being applied at the weakest portion of the skull, viz., the vault, the fissures should be widest apart at that point, becoming smaller and less widely separated as they reach the denser and stronger portions of such bones as the petrous; but the reverse obtains. In proof of my statement, examine this skull. Again, the case taken as my text and the two specimens that I show you here, besides other recorded cases, show that in the slighter cases of fracture of the base the vault is not fissured. Now, if fracture of the base does occur in these cases without fissure of the vault, severer ones, where the vault is so injured, I consider, can be caused in the same way; but, the force exerted being greater, the fissures starting at the base simply extend farther, and thus implicate the vault. The separation of the masto-occipital suture in the three cases cited—mine and these two in the Mütter Museum—seem to give the key to the mechanism

[1] The dense ridges of bone described are also actually amplifiers of vibrations.

of the severer fractures. It would be impossible to produce this effect if the fracture started at the vault, unless the upper continuation of the same suture were separated, and that, too, more widely than below. Presenting the appearances seen, the injury must have been produced by a spreading force acting somewhat in the way moistened beans do in separating a skull. If the fracture of the base started in the vault, the suture could be separated only by fracturing it so as to drive one bone below the level of the other; but this is *not* the appearance found. If, on the other hand, it commences at the base, the vibrations fracture it, and, displacing the bones, the lower part of the masto-occipital suture would be—as it is—*forced apart from within.* This is clearly the case in the specimens that I show you, and in the case related. I believe that this is *always* so, and that the appearance in the three cases cited is the key to the whole subject.

By examining the phenomena presented by contusions of the brain, an additional proof will be found of this conduction of vibrations to the points mentioned. Prescott Hewett states that the *under* surface of the cerebellum is the part of that organ most frequently contused; that the posterior lobes of the cerebrum, resting, as they do, upon the tentorium, are but seldom injured; that the anterior lobes are more frequently so; but that the middle lobes are vastly more frequently contused. He explains these facts by the relative frequency of fractures in the various regions and the numerous sharp, irregular projections of the cranial base, "which, although rounded off to a certain extent, and smoothed down by the dura mater, are still both sharp and numerous." Now, as contusion is frequently found in various portions of the brain *without any fracture*, and as even in fracture of the base the fragments themselves do not usually injure the brain, the only explanation left is to consider them as largely due to the conduction to that point of powerful vibrations. As to the statements concerning the sharp points, etc., I can only refute them by again calling your attention to the actual preparations before you, together with the position of the cerebro-spinal fluid. How Mr. Hewett and M. Fano can make such statements after examining a skull in the fresh state passes my comprehension. They have eminent authority, however, to fall back upon, whose want of observation still more surprises me, viz., Sir Benjamin Brodie, who says, "The great irregularities which exist on the inner surface of the basis of the cranium sufficiently explain wherefore the inferior is more liable to be ruptured than the superior surface of the brain." Mr. Hewett adds, "And nothing could be more ex-

plicit." It is only *explicable* to me by supposing that they examined—as is much too customary—only the *dried*, not the fresh, skull.

Let us examine President Lincoln's case, similar ones to which are explained by Longmore in the following manner: The lesions are to be ascribed to a "transmitted undulatory stroke or sudden impulse of the brain-substance itself against the thin bony layers constituting the orbitar plates. I am in possession . . . . of the notes of a case in which a similar fracture took place in one orbitar plate, from a ball passing along only grooving the upper surface of the hemisphere lying over the plate broken." Dr. Otis, as before said, accepts Prof. Longmore's opinion as being more satisfactory "than the hypothesis that the fracture was produced by *contre-coup*. The unusually thin orbital plates on either side were exposed to the impulse of the cerebral pulp. Even if they were not fully protected from the vibrations in the vault of the cranium by the dense supra-orbital ridges, it might be inferred that the force would be transmitted mainly to the right orbital region, or that opposite the entrance-perforation, whereas *both* orbital plates were fractured."

Dr. Allen also quotes Prof. Longmore's reasons for thinking that the shock could not be transmitted along the cranial walls to "the expanse of the frontal bone itself and the several processes within which the orbital plates are held, and by which they are so strongly protected in all directions laterally." As I have just said, my study of the cranium leads me to a very different conclusion with regard to Lincoln's fractured base from that of Dr. Allen. Let me quote his own words: "Now, in the case of President Lincoln, the ball, not expending its force about the wound of entrance, yielded the greater part to both tables of the cranium. The outer diploic layer appropriated the vibrations, and transmitted them to the great wings of the sphenoid bone, and thence to the axis of the cranium, without injury to the skull. The inner plate, not having any means of diverting its burden of force, carried it forward in two different lines from the wound of entrance to the orbital plates (which lie, as is known, about on a level with the upper margin of the tentorium), and broke them. It is to be inferred that, had the ball entered either above or below this line, the conduction of waves towards the forehead would have been greatly modified." How it is possible for the orbital plates to be protected from vibrations by the "dense supra-orbital ridges" I cannot see; nor why the "right orbital plate" should be more likely to be broken, except upon the old exploded theory of *contre-coup*, is not plain. Again, Prof. Longmore,

with Dr. Otis, seems to misapprehend a most important mechanical fact. If the force which breaks the orbital plates be of the nature of a something from without, entering between the particles of the bone and pressing them asunder, *then* the "expanse of the frontal bone," the "several processes within which the orbital plates are held, and by which they are so strongly protected in all directions laterally," and the "dense supra-orbital ridges," might perhaps avail. The truth is, however, that it is the vibrations of the bone itself which become so marked as to overcome the cohesion between the particles, and some, if not all, of the same dense bony ridges are the means by which these disrupting waves reach the orbital plates.

It is stating a mechanical impossibility to say that the vibrations were carried "forward in two different lines." As well might one expect a pebble thrown into water to send ripples in but two directions. The truth is, that the ball, entering one inch to the left of the median line, just above the left horizontal arm of the occipital cross, started vibrations which passed in *every* direction. If, however, the views are correct which I have just explained, the vibrations soon met with the thickened margins of the groove for the superior longitudinal sinus, which, being the best conductor and reinforcer of them, carried them to the frontal bone and crista galli. The usual damper of vibrations here was insufficient, and, they being propagated through the anterior fossæ, the weaker parts of the latter gave way. Doubtless, too, vibrations reached the orbital plates through the lesser wings of the sphenoid, and were coefficient in producing the fracture. On the other hand, those passing downward were safely conducted off by the means already indicated when speaking of the course of the vibrations.

A valuable fact pointed out by Prof. Allen is, that the occipital condyles really consist of two facets, often entirely separated from each other by a distinct groove, and he points out how if, at the time of force being applied, say from a fall on the feet, the head be extended, the force will affect chiefly the posterior facets, while if the head be flexed the anterior ones will have to bear the brunt. This probably explains, always subject to the laws of conduction of vibrations, why in one case a fall on the feet produces a fracture of the anterior and in another a fissuring of the posterior fossa.

Finally, we come to the reasons for the fatality of basal fractures. Of course, in the extensive tremendous crushes frequently found no explanation is needed. But recall to mind the cases I have related where the injury was so slight as almost to produce no symptoms at

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first; where the contusion of brain-substance, if such existed, was at a minimum; where, in my own case, no inflammatory symptoms were present. Here, clearly, some explanation is demanded. As long as such patients are quiet, as long as no vibrations are excited by the movement of locomotion, just so long the brain-substance contiguous to the lines of fracture remains uninjured. The primary jarring and slight contusions are recovered from; and, should quiet be sufficiently long maintained, recovery ensues. Let, however, the patient, tempted by his apparent recovery, leave his bed, immediately the normal vibrations which must mechanically arise are excited, and what follows? Before, in a state of health, they were safely conducted to points where they were annihilated; but now their course is interrupted by the fissures, and the superimposed brain-substance is jarred and probably slightly lacerated. From such constantly-recurring injuries, slight though they be, results, in the adult, meningitis or encephalitis, in the child, as in my case, very probably eclampsia, due to the greater excitability of the young brain. In proof of these slighter injuries induced by the slighter vibrations, let me again refer to the severer ones seen in extensive shattering of the base. As I then said, upon anatomical grounds, those found in the cerebellum and middle lobes of the brain cannot be produced to any great degree by direct injury from the fragments, but result from the tremendous vibrations conveyed to the brain when the bones give way.

It must now be clear that the anatomical facts demonstrated show: 1. That vibrations originated at any part of the skull do *not* pursue the most direct course, although they *do* follow the most direct anatomical route, to the base of the skull,—some, indeed, following all the anatomical courses, but the more distant more feebly, owing to the damping effect of the diploë and other tutamina of the brain. 2. That the means for rendering innocuous the ordinary jars of normal motions and many of the accidental blows, when the force applied is excessive, tend to the destruction of certain parts. 3. That fractures of the base much more commonly than is usually taught nowadays start *from* the base and extend thence into the vault. 4. That, when the normal route of the vibrations is interrupted by a fissure, those produced by the ordinary normal movement, then being prevented from terminating in their proper dampers, injuriously affect—perhaps even slightly lacerate—the brain-substance, and thus largely account for the greater fatality of such injuries over those of the vault. 5. That these facts should impress us with the necessity for the most rigid enforcement of

quiet in cases where we suspect or are certain of a fractured base, where quietude must be maintained for a lengthened period.

Dr. H. Lenox Hodge said that the paper presented by Dr. Nancrede was able, original, and instructive. Many of the conclusions would meet the hearty approval of all. He regretted that Dr. Nancrede had not completed his paper by performing and reporting a series of experiments upon the cadaver. The number of post-mortem examinations that can be obtained after fractures of the base of the skull is necessarily limited. The various forms of injury that produce fractures of the base of the skull can be readily repeated on the cadaver. The effects of blows, of falls, and of gun-shot injuries can thus be easily studied.

Clinical observations and experiments have brought out many points of interest. Injuries to the forehead are followed by fractures of the anterior fossæ, injuries of the temporal regions by fractures of the middle fossæ, and injuries to the occiput by fractures of the occipital fossæ. A fracture at the base of the skull is almost always accompanied by a fissure in the corresponding portion of the vault. The result of a blow or a fall on the head is due not only to the effects produced at the point of impaction, but also to those at the point of resistance. The cause of death after these injuries is due frequently to the direct effect of the vibrations upon the brain itself at the time of the injury, rather than to any secondary effects produced on it by the fracture of the skull.

Dr. Seiler said that he understood Dr. Nancrede to say that the injuries at the base of the skull were due to vibrations excited by the blow or fall upon the cranium, which were transmitted along the best conductors of vibrations, the thicker portions, to the base of the skull, where, by the meeting of different sets of vibrations coming over different routes, they were so augmented as to break the bone. He would like to call attention to the fact that this was an error of pathology, as the thicker portions of the bone were not better conductors of the vibrations than the rest of the base, but that they were capable of transmitting vibrations of greater amplitude. The conductivity of a substance for vibrations depended upon a proper relation of elasticity to density, and he cited as an example rubber, in which the elasticity is much greater than the density, and it is, therefore, a bad conductor of vibrations.

Dr. Seiler said that in regard to the second point there might be cases in which the vibrations might be dissipated by meeting instead

of their amplitude being augmented, and this took place, as he showed by a diagram on the blackboard, where the depression of one wave fell together with the elevation of another one coming from a different direction.

Dr. Charles K. Mills thought that in considering cranial fractures and concussion, and their consequences, the author of the paper should not have overlooked the investigations of Duret on Cerebral Traumatism. This distinguished cerebral physiologist has published the results of an elaborate experimental research and clinical study, under the title of "Experimental and Clinical Studies upon Cerebral Traumatism" (Paris, *Le Progrès Médical*, and A. Delahaye, 1878). The *American Journal of the Medical Sciences* for January, 1879, contains an able review of Duret's work. The cranium is a closed cavity with elastic walls. The elasticity of the skull can be readily proved. Duret mentions an experiment of Félizet, who filled a skull with melted paraffine, cooled it, and let it fall from a height of two and a half feet. The paraffine at the point corresponding to the blow showed a flat surface which indicated a depression of the skull of one-third of an inch. Not only is the skull elastic, but its contents are practically incompressible. A sudden blow on the cranium will, therefore, be transmitted to all parts within. A strong cask filled with a liquid may be burst by striking on the bung, or by pouring a few ounces of liquid into a long, vertical tube communicating with the interior of the liquid. Experiments of this kind are familiar both to practical men and to school-boys. Liquids transmit pressure equally in all directions, but, the contents of the cranium not being homogeneous, the effects of a blow upon the outside of the skull will be exerted from within outward upon certain regions of the brain and skull more strongly than upon others. The consequences both upon the brain and upon the skull are likely to be more disastrous when a blow or a fall is upon the top than when it is received upon the side of the head. Duret's explanation of this fact is that when the impact is in the transverse axis of the cranium a *cône de soulèvement*, or elevation of the skull, takes place at a point immediately opposite to the *cône de dépression* produced at the point of impact, and in this way the effect of the blow is diminished. When the blow or fall is received on the vertex, this saving cone of elevation cannot be formed, because the region opposite rests upon the spine and will not yield, and because also, perhaps, the structure and shape of the cranial base are such as largely to prevent such a result.

In cases of fracture at the base or at any other part of the skull, fatal

effects or serious results, such as fever, paralysis, or convulsions, are not always, and perhaps not usually, due to inflammation set up about the seat of fracture. The researches of Duret here again afford us a satisfactory explanation of phenomena which would otherwise be inexplicable. A blow or a fall sufficient to produce a fracture of the floor of the skull would be more than sufficient to cause the lesions and symptoms, either primary or secondary, which Duret studied under the head of cerebro-spinal shock. By blows upon the head and by injecting liquids within the skull lesions were produced in almost every portion of the brain. Some of these were the following: direct laceration and hemorrhages of the meninges; hemorrhages at various points at the base of the brain; extravasations of blood within the fourth ventricle; tearing of the foramen of Magendie; splitting of the floor of the ventricle; interstitial hemorrhages in the substance of the pons and medulla oblongata; hemorrhagic lesions even in the spinal cord. The symptoms produced by cerebro-spinal shock are, in very broad terms, changes in respiration, circulation, and intelligence, and spasm, tremor, paresis, or paralysis of certain muscles.

In regard to the spasmodic symptoms, in particular, which follow injuries to the base of the cranium or to other portions of the skull, Dr. Mills believed that at least three explanations might be given of their production: (1) direct irritation of the dura mater by spiculæ of bone, by clots, by spreading inflammation, etc.; (2) direct irritation by similar means of the cortical motor centres; (3) irritation from the effects of cerebro-spinal shock of the centres of the bulbo-pontine region. Numerous experiments by Duret have shown that the dura mater contains sensory nerves, readily excited, irritation of which gives rise to reflex spasms or contractures. The irritation is conveyed by these sensory nerves to the motor centres of the pons and medulla oblongata. Local irritation of the cortical motor centres may be caused by meningeal hemorrhages, by friction, and in other ways. By irritation from the effects of the cerebro-spinal shock he meant the rents, extravasations, and secondary inflammations of the convulsive zone of the floor of the fourth ventricle.

Dr. Mills also called attention to the analogy between the consequences of apoplectic shock and those of shock from blows and falls upon the head. In cases of hemorrhagic apoplexy the brain is struck a blow from within, instead of from the outside as in traumatisms. In sudden and extensive hemorrhages, displacement of the cerebro-spinal fluid sometimes takes place. He had recently reported to the Phila-

delphia County Medical Society a case of hemorrhage in the basal ganglia, followed by effusion of blood into the ventricles, in which the upper part of the floor of the fourth ventricle was found to be split to a slight depth in the median line. He thought it probable that at the time of the apoplectic stroke the cerebro-spinal fluid had been suddenly forced through the aqueduct of Sylvius, and that the walls of the fourth ventricle had been subjected to an outward pressure sufficient to cause a partial rupture. In such cases of apoplexy, besides the direct destruction caused by the effused and spreading blood, lesions similar to those seen after great external violence are usually present. These are small hemorrhages into and beneath the meninges, hemorrhagic spots in the pons, medulla oblongata and cord, etc. Many of the symptoms, immediate and remote, of apoplexy are the same as those of cerebral concussion or sudden cerebral compression.

In closing the debate, Dr. Nancrede, replying to Dr. Hodge, said that he could only again apologize for the many shortcomings of his paper, no one being more aware of them than himself. Unfortunately, however, owing to the absolute impossibility of giving more time to its preparation, the experiments that Dr. Hodge referred to had to be omitted. Dr. Hodge would bear him out in the statement that he had applied to him for some heads to experiments upon, but that time was wanting. He differed from him, however, as to the ease with which fractures could be produced. It is, indeed, a simple matter to cause an extensive fracture of the skull, implicating the base, but very difficult to produce one involving the base alone, as shown by the specimens and the case related, and which Dr. Nancrede contended give the key to the causation of the more extensive injuries.

With regard to Dr. Seiler's strictures as to the correctness of the term "conductors of vibrations," he acknowledged their propriety, and would, by his leave, correct it when publishing the paper. Dr. Seiler should recall the fact, however, that Dr. Nancrede in one part of his paper had termed the thickened ridges of bones "reinforcers" of the vibrations. As to the fact that the vibrations might meet and neutralize one another, he had thought that to be so self-evident as to require no statement regarding it. He would append a foot-note which would prevent others from being led astray by this omission.

Finally, as to Dr. Mills's criticisms, he must again say that his paper necessarily was limited to the consideration of certain points only. Perhaps a better title would have been "Some Hints as to the Etiology of Basal Fractures," rather than "The Etiology." He could

not, however, agree with Dr. Mills as to the explanation of the slight cases which he had distinctly stated formed the text for his paper. He acknowledged the truth of most of Dr. Mills's remarks, but still thought that the cases described were better explained by his own paper. Dr. Mills made no sufficient allowance for the displacement of cerebro-spinal fluid. Again, experiments do not explain the fact that the bony lesions do *not occur* exactly opposite the point struck, as the "cone theory" would imply, but at certain definite portions of the cranial base, to a marked extent irrespective (with certain limitations) of the point struck. Dr. Nancrede had, it would be remembered, not denied injury *to the brain* opposite to the point of impact. The cause of death, in the cases cited, did not seem to him due to the cerebro-spinal shock and its consequences, there being no evidences of active disease gradually supervening, from the traumatisms, *until normal vibrations had been excited in the cranial walls which failed to be conducted to their normal points for arrest, and in consequence irritated the brain and its envelopes.*

Again, Dr. Mills failed to appreciate the fact that when a fracture results the vibrations are chiefly, if not entirely, confined to the bone itself, it being a well-known fact that the more extensive splinterings of the vault, for instance, are often accompanied with but few evidences of injury and irritation of the subjacent brain: so that the constant contusion of certain portions of the base of the brain which are not necessarily opposite to the point struck, but correspond to the fracture, supports the views taught in the paper. *September 23d*, 1880.

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3. *Metastatic osteoid round-celled sarcoma of the skull, dura mater, and lower maxilla.*

Presented by Dr. Samuel W. Gross.

On the 27th of September, 1879, I amputated through the middle of the arm of a boy two years of age, on account of a periosteal osteoid round-celled sarcoma of the left ulna, which measured three inches and a half in length and two inches and a half in width, and began to develop at the age of three months. On dissection of the limb, four sarcomatous lymphatic glands, none of which exceeded the size of a large bean, were discovered along the line of the brachial artery above the elbow. In my comments upon the case before the Philadelphia

Academy of Surgery, October 6th, 1879,[1] I called attention to the fact that the ulna was rarely the seat of the disease; that the lymphatic glands were implicated in only 6.25 per cent. of the forty-five cases which I had analyzed; that the subject was the youngest on record; that the prognosis of these neoplasms was most unfavorable, since two-thirds of all patients die with metastatic deposits whether they are subjected to operation or not; and that the secondary growths were most common in the lungs, lymphatic glands, and the osseous system.

On the 25th of June, 1880, the child was brought to me with a densely-hard tumor of the right temporal region as large as the half of an ordinary orange. This continued to increase, but the child had been enjoying tolerable health, the only troubles having been complaints of the weight of the head and difficulty in mastication on the right side, on account of a tumor of the right ramus of the lower jaw, until the night of October 25th, when it was seized with convulsions, which were of short duration and very frequent, at least twelve having occurred in thirty minutes. These continued, at more prolonged intervals, until noon of October 27th, when death ensued after a violent paroxysm.

For the following notes of the post-mortem examination, which was made twenty-two hours after death, I am indebted to Dr. Ames, resident physician in the Jefferson Medical College Hospital:—

"Rigor mortis well marked; extreme emaciation; lower extremities œdematous; stump healthy. On the right side of the head there was a tumor, soft on its surface but hard at its circumference, which was limited above by the temporal ridge, and extended one inch below the condyle of the lower jaw. Its basal circumference was sixteen inches and a quarter, and it measured nine inches from the outer angle of the eye to the posterior portion of the parietal bone. The temporal veins were enlarged, and the skin was discolored just behind the ear. A second tumor was situated in the ramus of the right inferior maxilla, and measured three inches from the outer angle of the eye to the angle of the jaw, and three inches and a quarter from the angle of the mouth to the condyle of the jaw. Upon removing the firmly-adherent scalp, a tumor as large as a hickory-nut was discovered at the junction of the parietal and occipital bones, and was connected, through what seemed to be the posterior fontanel, with the interior of the skull. Another growth, of the same dimensions, was seated at the upper and

[1] Medical Times, July 3, 1880, p. 500.

posterior junction of the temporal and parietal bones. The left parietal, temporal, occipital, and frontal bones were of a dark-blue color, and the right parietal bone overlapped the frontal bone for a quarter of an inch. All the bones were thin and very brittle. The dura mater was congested, and an ante-mortem clot three inches long occupied the longitudinal sinus. The arachnoid was normal, but the pia mater was deeply congested. The substance of the brain was very soft, and its veins were engorged. A tumor three inches long, three and a half inches wide, and two inches thick occupied the middle fossa of the skull, and the corresponding lobe of the brain was almost diffluent. The tumor itself was firmly attached to the inner surface of the dura mater, and sent prolongations through that membrane to another tumor of about the same size growing from its outer surface. The large tumor of the right temporal bone appeared to originate in its medulla."

All of the smaller tumors are of a brain-like consistence, and do not appear to be the seat of ossific or calcareous deposits. The large tumor, however, is precisely similar to the primary growths of the ulna, and is beset by delicate spicules of bone which radiate from its centre to its circumference. As the specimens have been removed so short a time, there has been no microscopical examination of their minute structure ; but I have no doubt that they will be found to be of the same nature as the primary tumor, which consisted of small round-cells contained in a finely-granular matrix. *October 28th*, 1880.

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4. *Case of chronic peri- and intra-articular disease of knee-joint.*

Presented by Dr. C. B. Nancrede.

Peter ——, æt. 60 years, was admitted, September 13th, 1880, to the male surgical ward of the Protestant Episcopal Hospital, suffering from a slight but painful swelling of the left knee-joint. He had been a hard drinker and an inmate of the House of Correction until his transfer to my wards. He could assign no cause for his trouble, but said the other knee had been similarly affected and the disease had been pronounced rheumatic.

The joint steadily increased in size, but did not assume the peculiar form of acute synovitis, the swelling inclining to project more into the popliteal space and externally. By September 21, 1880, free fluctuation was detected, when the purulent collection was aspirated, and

proved to be distinctly extra-articular. The puncture soon ulcerated, when an exceedingly profuse discharge ensued, and continued for many weeks. After numerous counter-openings, oakum setons, etc. had been resorted to, the discharge diminished greatly in quantity, but the pain rather increased, until large doses of morphia were necessary to produce any relief. The joint became firmly anchylosed. During the month before operation he had two attacks of agonizing pain in the joint and its neighborhood, which so interfered with sleep and his appetite that he rapidly began to fail and was urgent to have his limb removed.

Accordingly, on January 17th, 1881, I amputated the thigh at the junction of the middle and lower thirds, after which he made an unusually rapid recovery.

Upon examination, the tissues surrounding the joint plainly indicated marked disease, while the joint itself was obliterated by adhesions, except over the under surface of the outer condyle, with the corresponding facet on the tibia, and over the patella, with the subjacent portion of the trochlear surface. Here there had been distinct hemorrhages, stretching and distending the adhesions. The inner facet of the patella was slightly eroded. It seems not improbable that the two sudden attacks of pain before referred to were due to the stretching of the adhesions produced by the sudden giving way of bloodvessels pouring out blood into the two small cavities just described.

Dr. Gross considered that in this case the trouble arose from inflammation of the lamella of bone just beneath the cartilage coating the patella. This is the usual way for joint-trouble to commence, except when the synovial membrane is diseased. He thought that the eroded cartilage was the source of the hemorrhage and the pain, rather than any stretching of adhesions, which he considered impossible by any passing hemorrhages such as these effusions must have been.

*January 27th*, 1881.

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### 5. *Case of alveolar sarcoma of the femur.*

Exhibited by Dr. C. B. Nancrede.

The specimen was removed by exarticulation at the hip-joint, in January, 1881, from a man aged 35 years. The duration of the case is doubtful, but it probably extends over a space of about two years. A little over three months back, spontaneous fracture occurred. The

tumor involves the lower half of the femur, the popliteal space, and apparently the tibia. Owing to the unfortunate decomposition of the specimen, nothing definite can be said about the deeper portions of the growth or the condition of the knee-joint. A loud aneurismal bruit was heard over all portions of the growth, which was of variable density. In the fresh state, upon dissection, the growth was strictly limited in front and laterally by the fibrous expansions of the quadriceps, but the posterior muscles were involved near the attachments. Section of some of the nodules revealed a growth which closely resembled normal brain-substance in consistence, etc. Microscopic sections were obtained from portions removed within twenty-four hours of the amputation, but as before said, the actual involvement of the tibia or knee-joint could not be determined, owing to decomposition having taken place. The patient did well for a few days, but died finally on the eleventh day after the operation. He worried and fretted so lest something *might* go wrong, that although the wound was well united, except at portions of its edges, a few drainage-openings, and its deepest parts, he actually seemed to die on account of his dread of death, which reduced his vitality to such a low ebb as to render him a ready victim to blood-poisoning, to which, although shown in no definite form, he seemed finally to succumb. *March* 10*th*, 1881.

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### 6. *Osteo-sarcoma of knee-joint.*

Exhibited by Dr. H. F. Formad, for Dr. C. A. McCall.

Mrs. R., 30 years of age, has four children, had two miscarriages, and one set of twins at full time. Weighed while in health about one hundred and fifty pounds. Never had had a day's sickness previous to this lesion; not a trace of any hereditary or specific disease in her family.

Mrs. R. during the Centennial year experienced, for the first, pain upon protracted exercise, in the right knee. A few months thereafter the leg would become suddenly flexed without apparent cause, thereby occasioning frequent falls, one of which, while stepping out of a carriage, resulted in a violent blow upon the right knee against the edge of the curbstone. Synovial inflammation at once set in, with all the usual symptoms,—pain, swelling, enlargement, fluctuation, etc. This condition becoming chronic, anchylosis to a considerable degree was established. In the latter part of 1877, Prof. Agnew broke up the adhesions, and the leg was placed in Stromeyer's apparatus; this was

borne but indifferently well, and before another six months the distortion was reproduced. At this time the swelling of the joint began to be more marked; pain was excruciating, but intermittent; the patient's health began to give way; she became thin and anæmic, and, though not strictly confined to her bed, showed all the symptoms of general "break-up." The case then left my hands, but came again under my care one year ago, when I found the leg in much the same condition as now presented in the specimen. Her general condition of health was bad, and in addition she was pregnant. A delay of nearly a year for any surgical interference was thus forced upon me.

November 27th, after the puerperal state had fully passed, and the patient being in good condition of nutrition, amputation was resorted to, and performed at the upper third of the thigh. She stood the operation well. The stump healed, and she is regaining apparent health.

At the time of the operation the tumor had reached the size of an adult's head. The skin was freely movable over the tumor, and no discoloration or ulceration was present. The lymphatic glands were not involved. The tumor was of white color, soft in consistence, contained numerous fragments of bone in its parenchyma, but was not enveloped in a bony capsule. The lower portion of the femur and parts of the tibia and fibula, as well as the patella, were involved in the new growth.

Microscopic examination revealed the typical structure of *alveolar sarcoma*, strongly intermingled, however, with the spindle-celled variety of sarcoma.

Dr. Formad said that, in addition to the information given in the notes, he would say that the patient had done well since the operation, having gained in flesh and strength. He thought that both Dr. Nancrede's case and his own were remarkable in this combination of round and spindle cells, which is certainly uncommon in such a situation, constituting the rare form of alveolar sarcoma.

Dr. Gross called attention to the rarity of alveolar sarcoma, and said that these two specimens were, he believed, the only ones that had ever been exhibited to the Society. In his lectures before the College of Physicians upon sarcoma of the long bones, he had then only one case of alveolar sarcoma to report, the specimen having been sent him by Dr. Burchard, of New York. He thought that, contrary to Dr. Formad's opinion, the outlook for Dr. McCall's case was very bad, as this form of sarcoma is peculiarly fatal. He would also call attention to the unusual fact that the knee-joint had been invaded and completely destroyed. This was the first instance out of many

examined by him where this condition had obtained. Extension of this disease from the tibia to the femur, or *vice versa*, is not uncommon, the morbid growth seeming to travel along the crucial ligaments. There seemed to be numerous portions of the specimen presented by Dr. Formad which were calcified. Dr. Gross would, therefore, class this tumor as a calcifying alveolar sarcoma. *March 10th*, 1881.

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7. *Specimen of necrosis of radius.*

Exhibited by Dr. C. B. Nancrede.

This specimen was removed from a married woman, 35 years of age, who three or four years since had her arm, as she calls it, "badly sprained." Some months after this the swelling and pain increased, suppuration ensued, and numerous sinuses formed. At the time of my examination, before admitting her to my wards in the Protestant Episcopal Hospital, the arm presented the ordinary appearance of necrosis with fracture either of the sequestrum or involucrum, as movement induced crepitus. At the operation it was seen that death of the whole shaft, for an inch or more, had occurred, that the necrosed segment had been nearly absorbed, and that its fracture had been the result of some trivial violence. The special point of interest was that nothing worthy of the name of involucrum had been formed, only a few nodules of osseous material being detectable in the surrounding periosteum. Condensing osteitis was present to a marked degree contiguous to the necrosed and carious bone, rendering its section very difficult. Dr. Nancrede thought that the specimen was of interest from the rarity of the failure of the periosteum, *in chronic cases* of bone-trouble, to form a marked involucrum. *March 24th*, 1881.

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8. *Specimens removed in a case of preventive trephining for a compound comminuted depressed fracture of the cranial vault.*

Presented by Dr. C. B. Nancrede.

Hugh McD., æt. circa 25 years, was admitted to the male surgical wards of the Protestant Episcopal Hospital in August, 1880. When I saw him his mind seemed clear, but his pulse was decidedly slow for a man who had been so severely injured, and who had lost considerable

blood. He had been struck down into the hold of a vessel by the falling upon him of about a ton of iron ore, which crushed his left leg to such an extent as to demand amputation. The fracture of the skull-vault must have been produced by his head striking against the corner of a bolt-head or nut, as it had evidently resulted from contact with some small, comparatively pointed object. The bone was much depressed over a small area, somewhat resembling a punctured fracture. Considering that, from its appearance, the inner table was probably extensively fractured, and believing as I do in "preventive trephining," I removed a circle of bone, elevated the depressed portions, and removed a number of fragments of the inner table, which had been driven down upon the brain and forced beneath the contiguous undepressed portion of bone. The wound was dressed antiseptically. As far as the head-trouble went, he did uninterruptedly well. If more compound comminuted depressed fractures of the skull were elevated before secondary mischief was lighted up, many more cases would recover than do under the present "let-alone" plan of treatment. I merely present this case as a typical one, for this is the second within a year in which I have performed preventive trephining successfully. After inflammatory trouble has been set up, trephining does not usually effect more than a temporary relief of the symptoms.

Dr. Henry asked whether any symptoms of motor paralysis had been noted, as such cases would tend to prove the correctness of Ferrier's views. The site of this injury was such as should have produced motor paralysis of the upper extremity.

Dr. Nancrede said that none had been detected.

Dr. Seiler related a case which he had seen when last in Vienna, where, by the knowledge gained by the experiments as to cerebral localization, the surgeon had correctly diagnosed the site of an old depressed fracture, which he had successfully removed by the trephine.

Dr. Henry mentioned Dr. Detmold's successful case of trephining and opening of an abscess deeply seated in the brain-substance.

Dr. Nancrede then made some remarks upon what had been done in the direction of a practical application to surgery of cerebral localization in France, and cursorily reviewed some of the recent literature of the subject. *June 22d*, 1881.

## II. THE DIGESTIVE APPARATUS.

1. *Hepatic abscesses from a case in which there were cystic ovarian tumors containing inspissated pus.*

Presented by Dr. Louis Starr.

I am indebted to Dr. Sinkler for the opportunity of presenting these specimens. The clinical notes were taken by Dr. H. D. Harvey, resident physician at the Episcopal Hospital.

Kate ——, æt. 35, was admitted to the medical ward of the Episcopal Hospital on August 16th, 1879. Her family history was good, all her immediate relations being alive and well except her father, who had died of dysentery. She had always resided in Philadelphia, had been temperate in her habits, and had never had syphilis. Exclusive of the diseases incident to childhood, she had enjoyed good health until the commencement of her last illness, in June, 1878.

At that time she noticed a small, painless tumor in the left iliac region, her appetite failed, she had considerable flatulent distension after eating, and her bowels became constipated. The tumor slowly increased in size. In May, 1879, the catamenia, which had previously been regular, stopped, and she began to have tensive pain in the tumor varying considerably in intensity, to lose flesh and strength, and to suffer from neuralgia of the left sciatic nerve occasionally severe enough to prevent her walking.

Early in August she noticed a dull pain in the right infra-mammary region, and on each of the two days preceding her entrance to the hospital she had several copious evacuations from the bowels. Nevertheless, she continued to perform her household duties up to the day of admission.

When admitted, she was very weak, her face was anxious, the conjunctivæ were slightly yellow, and the skin somewhat sallow. Her tongue was covered with a thick, brownish coating, which was dry and cracked; she had little desire for food, and there was moderate diarrhœa, the evacuations being liquid, but containing neither mucus nor blood. The abdomen was distended; the left iliac, the hypogas-

tric, and the umbilical regions were occupied with a tumor, which was dull on percussion, tender to the touch, and gave to the finger a sense of firmness, with indistinct fluctuation; the remaining regions were tympanitic. Her breathing was hurried, and there was dull pain in the right infra-mammary region, uninfluenced by the respiratory movements. The pulse was frequent and feeble.

On August 17th and 18th, the pain in the tumor became greater and the abdominal tenderness more extended.

On August 19th, the pulse was 120 and very weak; there was flushing of the face and considerable elevation of the surface temperature, followed in the afternoon and evening by profuse sweating. During the night there were two large evacuations from the bowels, consisting of little else than dark-colored, clotted blood.

On the morning of the 20th the patient complained of great pain in the lower portion of the abdomen, her skin was cold and clammy, and her pulse thready. In the afternoon there were two more hemorrhages from the bowels, and death occurred early in the evening.

An *autopsy* was made fifteen hours after death. The body was much emaciated. On opening the abdomen, a large mass was observed filling up the lower portion of its cavity and displacing the intestines upward and to the right. This mass in reality consisted of two tumors bound together by narrow bands of fibrous tissue. Both tumors were lobulated and fluctuated indistinctly under the finger. The smaller of the two occupied the left iliac fossa and the left iliac region, was about four inches in diameter, and was loosely connected with the left side of the body of the uterus; the larger occupied the hypogastric and umbilical regions, was about five inches in diameter, and was more intimately joined with the fundus and the right side of the uterus. The dilated right Fallopian tube could be traced along the under surface of the larger tumor, and the left Fallopian tube could be traced for a short distance into the smaller tumor. Upon section, the tumors were found to be cystic in structure; their walls were about one-eighth of an inch in thickness, and they contained a soft, putty-like substance resembling inspissated pus, the material filling the larger sac being white and firm, while that in the smaller was more liquid and yellowish in color. The uterus appeared to be normal. The tumors, together with the uterus, weighed a little more than seven pounds.

The *liver* was somewhat enlarged, the right lobe extending from the costal border upward as high as the fourth rib, and the margin of

the left lobe being too far to the left. The upper right postero-lateral surface of the right lobe was adherent to the diaphragm and abdominal wall, and considerable force was required in removing the viscus. In doing this the hepatic tissue was torn, opening a large abscess situated superficially in the upper posterior part of the right lobe, and giving vent to a quantity (estimated at one pint) of inodorous, thick pus, containing numerous shreds of partially broken-down glandular tissue. The abscess measured six inches in transverse and five inches in antero-posterior diameter; its posterior wall was scarcely one-fourth of an inch in thickness, and its internal surface was ragged, being formed of softening tissue. A short distance behind and below this abscess was a second abscess, entirely isolated, irregular in shape, and about as large as a walnut. The substance of the liver in the neighborhood of the abscesses was very much softened and engorged with blood. The gall-bladder held a small quantity of viscid, bile-stained mucus; its walls were thickened in an appreciable degree, and the reticulation of the mucous membrane was unusually marked. The bile-ducts were patulous. The liver weighed five and a quarter pounds.

The stomach and intestines were not examined.

The kidneys and the thoracic viscera were healthy.

A microscopic examination made by Dr. Simes showed that the material contained in the ovarian cysts was composed of pus-corpuscles, granular débris, and compound granular corpuscles.

*Remarks.*—There can be no doubt that the abscesses in this case were pyæmic in nature, although the anatomical characteristics of pyæmic abscesses of the liver, viz., multiplicity and smallness in size, were absent. The absence of these characters is, I think, not unusual; for example, in three other cases[1] which have come directly under my observation there was in one a single large abscess in the right lobe, in another a large abscess in the right lobe and a small forming abscess the size of a filbert in the left lobe, and in the third there were two abscesses in the right and one in the left lobe, the largest being five inches in transverse and six inches in antero-posterior diameter, and the smallest about an inch in diameter.

In this patient, in addition to the suppuration in the cystic tumors—a very evident source of purulent infection—there was probably some lesion, perhaps ulceration, of the intestinal tract, though unfortunately this was not investigated at the post-mortem examination. That a

[1] Transactions of College of Physicians of Philadelphia. Third series, vol. iv.

lesion did exist I infer from the diarrhœa and intestinal hemorrhage which occurred a short time before death, the color of the blood showing that it came from a point rather high up in the canal, and also from the fact that there was no evidence of portal obstruction or of pulmonary or cardiac disease to cause extreme congestion of the intestinal mucous membrane and consequent hemorrhage.

From a clinical stand-point the case must be placed in the large group of *latent* hepatic abscesses, there being no symptoms indicative of disease of the liver except the slight yellowness of the conjunctivæ and the dull pain in the right infra-mammary region, neither of which is at all characteristic. On the contrary, the symptoms, and especially the physical signs, were such as to fix the attention upon the abdominal tumor. *September 11th*, 1879.

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2. *Case of obstruction of the bowels where laparotomy was performed.*

Presented by Dr. Nancrede.

George Ott, male, æt. 29 years, by occupation a brickmaker, was admitted to the male surgical ward of the Protestant Episcopal Hospital on the afternoon of August 19th, 1879, suffering from a horse-kick in the lower part of the hypogastric region.

The pubic region was much ecchymosed, one calk of the shoe having struck in the left inguinal canal, and the other about one inch above this spot on the right side. He had never suffered from any hernial trouble. Symptoms of mild peritonitis supervened, which were controlled by the administration of eight grains of opium per diem, with the exception of occasional vomiting when the stomach was overloaded. On the afternoon of the third day, however, the ejecta smelt stercoraceous, and by the following morning he was collapsed with undoubted stercoraceous vomiting. He now presented a swelling in each inguinal region. That on the left side receded completely when taxis was used, but with an emphysematous crackle unlike the characteristic "gurgle" of reduced gut. That on the right side could not be entirely removed by taxis, leaving a small mass which felt like a hernial sac.

After consultation, it was determined to cut down upon the right inguinal canal, and endeavor to discover the nature of the irreducible part. When the peritoneum was reached, it was black and sphacelated in parts, so that during some of the manipulations it gave way, giving vent to several ounces of stercoraceous-smelling matter. No further

search for the obstructed portion of the gut was undertaken, owing to the matting together of the bowels by adhesions and the apparent demonstration of the rupture of the bowel.

Death took place seventeen hours later, and on post-mortem examination no obstructed portion of bowel was found, water passed freely through the gut, and no ante-mortem rupture could be discovered. At several points the whole circumference of the gut was inflamed throughout all its coats, thus giving an illustration of what is more common than is usually suspected, viz., an obstruction of the bowels from inflammation of all its coats, thus producing paralysis at that point. The fecal odor was probably due to the osmosis of the intestinal gases into the purulent peritoneal fluid.

*September* 11*th*, 1879.

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### 3. *Carcinoma of pyloric extremity of the stomach.*

Presented by Dr. J. C. Wilson.

This specimen was removed from the body of a man, 56 years of age, who was admitted to the medical ward of the hospital of the Jefferson College on the 15th of November, 1879, and died six days after.

He stated that his health had been good until the winter of 1878–79, when he had several attacks of cold and lost strength, which was never regained. During the winter he was dyspeptic, and began to suffer pains in the epigastric region, most intense some time after eating, a sense of weight, belching, occasional vomiting, which never contained blood. These symptoms continued until two months ago, when the vomiting ceased, and the pain became less irksome.

Has had several attacks of faint jaundice, habitual poor appetite, constipation, with occasional but never long-continued white stools.

A few weeks before he came to the hospital he became dropsical.

On admission, there was extreme universal anasarca, with evidence of large abdominal and moderate pleural and pericardial effusions. There was slight tenderness of the belly on palpation. The respiratory murmur was faint, and not well heard through the thick and œdematous chest-walls. Heart-sounds feeble; at apex a faint systolic murmur; the second aortic sound not relatively intensified. Liver dulness in mammary line and in the line of the anterior axillary fold

not decreased. The urine, on admission, contained no albumen; later, a trace.

At the autopsy, the heart was found enlarged, fatty, its muscle-substance the color of a faded leaf, mottled and streaked. The coronary arteries were rigid and highly degenerated. The leaflets of the mitral valve were thickened; those of the aortic valves and those of the right heart showed no morbid change. Kidneys small; the cortex diminished; capsules adherent. The stomach was found to be the seat of a tumor situated at the pyloric extremity, cylindrical in shape, measuring transversely two inches and longitudinally three inches. The opening into the duodenum remained patulous, admitting the finger without difficulty, which is astonishing considering the size of the tumor. An examination of the other viscera showed them to be free from secondary deposits. *January 8th*, 1880.

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### 4. *Perforation of the appendix vermiformis.*

Presented by Drs. Eskridge and Mills, for Dr. J. C. Norris.

Mr. J. F. D., aged 53 years, left Batavia, New York, in good health, on Thursday, September 25th, 1879, for Bethel, Connecticut. After his arrival at the latter place he partook of a luncheon of cheese and grapes, and soon afterwards was seized with severe pain in the hepatic region. He was attended by Dr. A. E. Barber, who has kindly furnished a brief statement of his experience with the case. Dr. Barber says that he was first called in to see Mr. D. on Friday, September 26th. He found him suffering pain in the hepatic region, with a little tenderness over the abdomen. Morphia was administered hypodermically, and gave relief. He was more comfortable on Friday evening, but his tongue was somewhat coated, his pulse was 115, and he had some fever. On Saturday morning he seemed better; his pulse was 100, and he still had some fever. A fever mixture and morphia and camphor-water were ordered. Saturday night he was restless and had considerable pain, but slept most of the time until Sunday morning. On Sunday, Monday, and Tuesday he became more comfortable each day, and, as he was up and around on Tuesday afternoon and on Wednesday, Dr. Barber reluctantly consented to his leaving for Maryland, recommending him to wait a day or two longer. As he felt comfortable, however, he concluded to go as far as New York city on

Wednesday. He reached that city without special inconvenience or discomfort, and on Thursday came in a Pullman car to Philadelphia. On his arrival he refused to take a carriage, but rode in a street car to No. 921 North Sixteenth Street, where he intended to stop for a short time. He took a bath and ate his supper, and afterwards Dr. J. C. Norris was called in to see him. Dr. Norris found that he was not suffering much. He complained of slight pain in the hepatic region, but had no cramps and no abdominal tenderness. His pulse was 120, feeble, and occasionally intermittent; his tongue was brown and dry; his extremities were cold, but the doctor thought that he had not reacted thoroughly from his bath. He prescribed opium, digitalis, and camphor; and whiskey, beef-tea, and milk were ordered. Dr. Norris was sent for at five o'clock the next morning (Friday). He found him cold and pulseless; he continued to sink, and died at seven A. M. His wife stated that he had been on his feet at one o'clock, and after this seemed to sleep well, but at four o'clock she found that he was cold. He complained of nothing during the night.

The autopsy was held twenty-four hours after death. Muscles rigid and body well frozen. All the tissues appeared well nourished. Fat was abundant, being an inch thick over some portions of the body. The thoracic and abdominal cavities only were examined.

*Thorax.*—The lungs were apparently healthy. The heart was nearly normal; slightly hypertrophied, and well supplied with fat, but no evidence of any of the muscular fibre having undergone fatty degeneration. All the valves and orifices were normal. Beginning ossification of the walls of the ascending portion of the arch of the aorta was present.

*Abdomen.*—The walls were very thick from the abundant deposit of fat. The entire peritoneum was more or less injected, and the lower portion, especially that on the right side, highly inflamed. Pus and lymph filled Douglas's cul-de-sac, and a large portion of the right iliac fossa. The bowels, peritoneum, and walls of the right lower portion of the abdominal cavity were intimately adherent. Those portions of the bowels and peritoneum which lie in this region of the abdomen were so closely glued together that the pus formed in their interspaces was unable to escape, a large fluctuating substance like an abscess of huge size being thus formed. When the adhesions were carefully torn away, over half a pint of pus was found in the tumor-like mass. The bowels immediately above and below the vermiform appendix were highly inflamed, darkened, and softened, with the

mucous surface undergoing the suppurative process. In the cul-de-sac of Douglas, after repeated searches for some offending substance, a cylindrical body three-fourths of an inch long and one-fourth of an inch in diameter was found. Sections of this, under the microscope, showed it to be composed of fecal matter.

This case, like those of a similar nature previously reported to this Society, well illustrates the importance of keeping the patient and his bowels perfectly quiet for a length of time when any trouble in the region of the appendix is apprehended. Had this man remained quiet for a week or two, suppuration, if it had already begun, might have been arrested, or, if perforation had taken place, death would possibly not have resulted, because adhesions had already formed around the region of the appendix sufficient, if the recumbent posture had been maintained, to prevent the general diffusion of pus, which gives rise to shock. Cases are on record, and some have occurred in the practice of members of this Society, in which recovery has taken place although all the symptoms of inflammation and suppuration in this region have been present. In these, after recovery, a large tumor has been found in the right inguinal region. *January 8th*, 1880.

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### 5. *Carcinoma of œsophagus and stomach.*

Presented by Dr. L. S. Clark, for Dr. Newcomet.

Captain N., æt. 54, robust, florid complexion, a spirit-drinker; employed as captain in merchant marine; during the war pilot on transports; since the close of the war a hotel-keeper, but for the last few years again on water as pilot, his last service being on Fairmount boats.

About August 20th he called at my office, complaining of, as he called it, *acid dyspepsia;* he had an acid taste in his mouth, with vomiting, but not much pain. He still kept at his post, but began to lose flesh and strength, the distress becoming more marked, with difficulty in swallowing, which increased very rapidly. Solids were entirely discarded; even rice and barley could not be taken; milk was taken for some time, but frequently vomited; always complained that it hurt him; beef-tea and milk would "come up," as he called it, while water did not distress him so much.

During the act of deglutition he placed his hand over the lower part

of the œsophagus, saying, "Here it is," and in a few moments he would regurgitate, apparently by spasmodic action. No nourishment could be taken by the mouth for nearly two weeks, during which time he complained of pain over the cardiac region. The family record was good, so far as cancer was concerned.

The post-mortem showed an extensively diseased condition of the stomach and œsophagus. *January 8th*, 1880.

*Report of the Committee on Morbid Growths:*

"The specimens were so far advanced in decomposition that it was impossible to make a histological examination of them.

*January 22d*, 1880."

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### 6. *Carcinoma of the œsophagus.*

Presented by Dr. L. S. Clark, for Dr. Newcomet.

P. R., æt. 57, a robust German, in this country twenty years; surrounding circumstances (locality, etc.) fair; habits very fair.

About four years ago he began to notice that while drinking, especially beer or wine, there would be regurgitation (it would come up without an effort, as in emesis). He was frequently accused of intoxication on that account when he had drunk nothing.

On May 4th, 1869, he met some friends in a wine-house, when the above-named trouble was observed, and he was advised to call on me. I was convinced there was stricture of some kind. Professor Agnew examined him about a week later, and verified my diagnosis. On swallowing he made a double effort. By applying the ear to his chest along the lower part of the œsophagus one could hear a second sound as produced by swallowing.

Solids were rejected at once. Semi-solids could be taken with great difficulty, only a little at a time and at long intervals. He soon became unable to swallow anything but liquids, and even beef-tea could not be taken. Wine and water, a teaspoonful at a time, at intervals of about ten minutes, were retained. At last he would take nothing but ice. Strange to say, he had no craving for food.

He was very much emaciated; had no pain, and was hopeful to the last. Died without a struggle, purely from exhaustion, on July 18th, 1869.

On post-mortem examination we found all the organs in comparatively good condition, except the œsophagus, where we found the tumor submitted to you for examination. *January 8th*, 1880.

*Report of the Committee on Morbid Growths:*

"A section made from the diseased portion of the œsophagus presented by Dr. Clark is found, upon microscopical examination, to have its mucous surface in greater part destroyed by ulceration. At the margin of the ulcer the mucous membrane is seen to have an increase of the laminated epithelium, the inter-papillary processes being very much enlarged, branching, and extending into the submucous tissue; also in this latter tissue are observed cylindrical collections of epithelial cells, separated by a highly-inflamed fibrous connective tissue; the new formation is a squamous epithelioma.

*January 22d*, 1880."

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### 7. *Carcinoma of the stomach.*

Presented by Dr. Morris Longstreth.

I am indebted to Dr. Thomas George Morton for the specimen and the history of the following case:—

The patient was an Israelite, æt. 56, had long been engaged in a manufacturing business, and had always enjoyed good health. He consulted Dr. Morton on April 22d, 1879, on account of certain vague symptoms, which perhaps can be best summarized by saying that the patient was not in his usual good health, and that his digestion was poor. The patient passed a very large amount of urine daily, and an examination showed its specific gravity to be 1033. Several careful tests with Fehling's solution for sugar resulted in an uncertain reaction. The fermentation test, however, showed a marked diminution of specific gravity of the urine, with the development of carbonic acid gas after the completion of the process. The diagnosis was made at first of diabetes mellitus, the irritability of the stomach being due to this cause. About a month later the patient began to reject his food at irregular periods. The vomiting usually did not occur until a number of hours after the reception of the food into the stomach; the patient would frequently eat his dinner and supper and not vomit until the following morning. By the beginning of June the increased frequency

of the vomiting, together with a change in the general aspect of the patient, indicated that the patient was suffering from some organic disease of the stomach. The gastric symptoms were confined to a feeling of distress and the vomiting; there was no pain, no sense of fulness or hardness on palpation of the epigastrium, and no vomiting of blood and no bloody stools occurred. During June and July Dr. Morton did not see the patient, being in Europe during this time, but on his return, in August, he found the patient vomiting much more frequently and regularly after taking food. At this time a tumor could readily be felt in the epigastrium; the patient was greatly emaciated and presented a markedly cachectic appearance. It was now no longer doubtful that the illness was due to malignant disease of the stomach. The gastric distress, constantly present, remained the same, the inability to retain food greatly increased, and the emaciation steadily progressed, and with it the deepening of the cachectic appearance. From September until the occurrence of death, on December 30, 1879, all attempts at effective nourishing by the mouth had to be abandoned, and resort was made to nutritive rectal injection. This therapeutic resort was regularly carried out thrice daily. The nourishment consisted usually of an egg beaten up with a small portion of brandy and milk; the injections were well borne by the rectum, and the hard, dry fecal mass was voided regularly every day. During all the latter period of life the only material given by the mouth was a little champagne. Late in life dropsical swelling of the legs and abdomen came on, but the emaciation of the body towards the last became extreme, and death resulted from inanition, without further development of symptoms in connection with the stomach. The glycosuric condition of the urine remained as first discovered for some time, but it gradually diminished, and finally disappeared before death. In connection with this symptom I wish to call attention to the involvement of the pancreas in the malignant disease.

The specimen shows a mass composed of the spleen, the stomach, a portion of the liver, with the gall-bladder, and the pancreas. The spleen is firmly attached on the posterior surface of the cardiac extremity. It is much reduced in size, its consistence is firm, and its capsule wrinkled, but, upon section, its tissue appears normal. The pancreas closely adjoins the spleen, and is firmly attached high up on the posterior surface of the stomach. At the lesser curvature of the stomach the liver is firmly attached by a mass of fibrous tissue con-

taining greatly enlarged lymphatic glands. The gall-bladder, which appears about normal size, overhangs the duodenum, just below the pylorus. The stomach, which can hardly be recognized as such, either from its shape or texture, looks like a thick-walled tube which is sacculated at one or two places. Its extreme length, measured in a curved line, is eight inches; its calibre, about that of the undistended transverse colon. Its walls are greatly thickened, measuring half an inch at the pylorus, and about one-quarter of an inch at the middle portion of the greater curvature. The tissue is as firm and rigid as the stoutest sole-leather, and the glands on both its curvatures are greatly enlarged and softened. The peritoneal coat shows numerous very small nodular elevations, and the general surface is somewhat roughened by inflammatory lymph. The muscular coat of the œsophagus at its entrance is very considerably thickened. Its mucous membrane is greatly thickened, tightly adherent by submucous connective tissue, and is thrown up into very prominent longitudinal folds.

The mucous lining of the stomach seems to have entirely disappeared, except at a small portion at the cardiac extremity.

The transverse section of the wall of the stomach shows the peritoneal coat to be greatly thickened; the muscular coat to be infiltrated with new material, which is seen to be traversed by bands of white fibrous tissue. At all portions of the transverse section of the wall it can be distinctly seen that the ulceration has not extended through the submucous connective tissue, and that the bands of fibrous tissue traversing the infiltrated muscular layer run from the submucous tissue to the peritoneal coat. The mucous-membrane surface of the stomach is everywhere roughened and presents a granular surface.

At some portions, especially at the posterior surface, it is evident that the ulceration has extended into the muscular layer.

A small portion of the liver, attached to the stomach, presents one or two small bean-sized nodules of whitish color, which appear as infiltrations into the fatty-looking liver tissue. One or two large nodules were found in other portions of the liver. The tail of the pancreas presents a large, rounded, nodular mass, which, on section, shows several white, softened, rounded masses. The other portion of the pancreas, on section, shows almost none of the characteristic appearances of the organ; the tissue is pale, and the fibrous tissue is greatly increased, and its ducts appear to be dilated. The head of the organ is involved in a mass of inflammatory lymph, in which are numerous

cancerous nodules, perhaps lymphatic glands, perhaps nodules in the tissue of the organ itself.

Just beyond the pylorus, the small portion of duodenum attached to the specimen shows very marked changes of its mucous membrane, with some ulceration. Just within the pylorus the ulceration is very deep, and has removed nearly the whole of the infiltrated wall.

*February 26th*, 1880.

*Report of the Committee on Morbid Growths:*

"A microscopical examination of a thin section of the stomach shows the new formation to consist of somewhat large alveoli containing large epithelial cells. The fibrous tissue constituting the alveolar walls is relatively small in amount. The neoplasm is an encephaloid carcinoma. *March 25th*, 1880."

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### 8. *Chronic gastritis, with dysentery, etc.*

Presented by Dr. Morris Longstreth.

The patient, Wm. H. C., painter by trade, was admitted to the Pennsylvania Hospital, August 9, 1877. He had contracted syphilis three years previously, and the primary lesion was followed by secondary manifestations; of this disease he appeared to be well, and the illness from which he suffered on admission to the hospital was not connected with its results. He appears to have suffered considerably from diarrhœa at various times; he had been in the war of the rebellion and in the swamps of the Chickahominy (but had never had malaria); he had dysentery while in the army in 1864. About three years before admission he was caught between a wagon and a train of cars, and received a contusion of the abdomen, which confined him to bed for six weeks. Two years ago he had an attack of fever, while in New Orleans, characterized by pain in his body and by delirium; the illness lasted about four weeks, but was not accompanied by diarrhœa. Since then he has never felt quite well, and three months subsequently he noticed that his skin was sallow, and he felt dull and drowsy. Six weeks before admission he was attacked by diarrhœa, without any known cause, and shortly after this his legs began to swell.

On admission he appeared emaciated, of a dirty, sallow complexion, but the eyes did not seem especially discolored. He was having six

or seven passages daily, loose in character, but not watery, slimy, or bloody, and there was no tenesmus, but occasional cramps in the abdomen. Tongue not coated, but pale and smooth. The splenic dulness did not extend to the margin of ribs. The liver was slightly increased in size. The gums were pale, but not spongy. The urine was normal. His general condition remained about the same for several months; generally confined to bed. The diarrhœa at times improved, and then again relapsed. In the latter part of November, 1877, the diarrhœa had become pretty constant and exhausting, and continued in spite of treatment and regulated diet. Towards the latter part of the time his appetite, which had been pretty good, failed. There was no nausea or vomiting, but he occasionally complained of obscure epigastric soreness, and there was a feeling of obscure resistance in the epigastric region, but the position did not correspond to the locality of his sense of soreness; there was no dulness on percussion over the abdomen, and there was no complaint of positive pain.

*Post-mortem examination*, by Dr. Longstreth.—Rigor mortis moderate; emaciation extreme; œdema of lower extremities.

*Cavities.*—The *abdomen* contained a considerable amount of serum; there was no evidence of recent inflammation. The omentum was rolled up over the stomach, and was tightly adherent.

Both *pleural* sacs showed some old fibrous adhesions, and the cavities contained considerable clouded serum, with clots of fibrin floating in it. No evidences of inflammatory action.

The *pericardium* was normal.

*Heart.*—Right side relaxed, but contained only a small amount of fluid blood and a few small clots. Left ventricle firmly contracted and empty. The organ was reduced in size; its orifices, valves, and cavities showed nothing abnormal. Its tissue was moderately firm and evidently atrophied, but not fatty. Weight, seven ounces avoirdupois.

*Lungs.*—These organs showed nothing especial to note.

*Spleen.*—It was of small size; its capsule was wrinkled and pale; consistence flabby, and its tissue anæmic and moderately resistant. Weight, five ounces avoirdupois.

*Kidneys.*—Both were small, pale, moderately firm, and slightly irregular in outline, but presented no special morbid change. Weight, four and a half ounces, left; four ounces, right.

The *liver* was small, regular in outline, pale, and anæmic; its capsule was roughened by a few fibrous adhesions; its tissue was evidently

atrophied, giving the appearance of a relatively greater amount of fibrous tissue, but there were no indurative or cirrhotic changes. Weight, two and a half pounds.

The stomach presented the appearance of a rigid, thickened-wall sac, having a length of six and a half inches; it is two and a half inches wide at the cardiac extremity, and one and a half inches wide at the pyloric end. Its peritoneal surface is rough, especially on the anterior surface, along the greater curvature where the rolled-up omentum was attached. At the posterior part of the cardiac extremity the organ was so firmly adherent to the part behind that its wall was torn in removing it. The only portion of the stomach that has a normal feeling when handled is a small portion just at the pylorus.

On opening the stomach its mucous membrane presented a condition of ulceration throughout nearly its whole extent, and its walls throughout their whole circuit were enormously thickened. At the œsophageal opening the muscular tunic was greatly thickened, but the mucous membrane was smooth and normal-appearing. At all other portions, excepting near the pylorus, the whole internal surface presented a rough, somewhat shaggy aspect; at parts the ulceration had extended in depth to the muscular layer, the fibres of which were distinctly visible; at other parts the surface was covered with shreds of the mucous or submucous coats. The edges of the ulceration, both towards the œsophageal opening and the pyloric extremity, presented thickened, everted borders, composed of the epithelial layers.

A small portion of the mucous-membrane surface at the pylorus showed the rugæ as in a healthy stomach, but its epithelial covering appeared to be to a considerable extent removed; however, the surface was smooth, and there was no appearance of ulceration.

A vertical section of the stomach-walls showed enormous thickening of all its coats, measuring in places half an inch. The thickening especially affected the muscular tunic, but there was great increase of the outer coat, and also of the mucous and submucous layers, except, of course, where the ulceration had destroyed the inner lining. From the fibrous and peritoneal coat, bands of connecting tissue could be seen, on the surface of a section, dipping down and passing in between the bundles of muscular fibres. So great was the increase of the muscular tissue that there appeared to be many layers of muscles, instead of the normal number.

The *lymphatic glands* along the lesser curvature of the stomach

were found enlarged and swollen, homogeneous on section, but not softened.

The *duodenum* directly without the pylorus appeared nearly normal, but beyond this its walls were thickened; its peritoneal coat was roughened by fibrous inflammatory tissue, and its mucous membrane in a state of catarrhal inflammation, but not ulcerated.

The *small intestine* showed throughout its length the results of a former morbid condition of its mucous membrane, especially thickening of its walls.

The *large intestine* was very greatly thickened, and in places contracted. Its mucous membrane showed evidences of old and deep ulceration; but in most places it was healing, and presented a smooth, board-like aspect.

The *mesenteric glands* were found much enlarged.

The *gall-bladder* contained but little bile, which was of a very pale color, and contained considerable mucus.

*Microscopic Examination of the Stomach-wall.*—The changes in the structural elements of the tissue are so great that it requires considerable study to identify them and to distinguish the several layers.

Commencing at the outer surface, the peritoneum is found to be covered by a *layer of fibrous material*, varying in thickness at different parts. This material presents a rough, shaggy surface; it is well organized, and shows numerous very small capillary vessels passing between its fibres; at parts there are seen very many indistinct cellular elements. It is firmly attached to the peritoneum, on which it is placed.

The subperitoneal tissue, which can be distinguished by the numerous vessels, seen mostly in cross-section, in it, is very much thickened. Its fibres are firm and coarse, and closely matted together. The vessels in this part are larger than usual, more numerous, and their walls thickened, and large lymphatic vessels are to be seen in it containing granular cells or nuclei.

Next to this coat comes the muscular tissue, in which can be seen layer after layer of alternate bands of muscular fibres, presenting first the ends of their cells cut in cross-section, and then running parallel to the length of the fibres; again, in other parts the muscular bands are seen running in oblique directions. The bands of muscular fibres are often widely separated by layers of connective tissue, and occasionally this connective tissue is visible passing in a perpendicular direction through the muscular bands. That it is not only an increase of the amount of muscular tissue, but also a true hypertrophy of this tissue,

is evidenced by the multiplication by division of nuclei of the muscular fibres, which can be seen occasionally in some parts of the section where these fibres have been cut transversely. Quite a number of vessels can be seen, some cut transversely, some longitudinally, running in various directions in the muscular-fibre layer. These vessels are frequently seen surrounded by clusters of small cells, which stain deeply, and are also accompanied by or have in their neighborhood lymphatic vessels, filled with brightly-stained cells of the same character.

The limitations of the muscular and submucous layers are well marked in nearly all parts of the sections by an expanse of considerable breadth of much coarser fibrous tissue.

The submucous layer is clearly to be recognized by the numerous large-lumened, thick-walled arterioles seen in it. It is composed of coarse-fibred connective tissue, which twists and sweeps in bands of varying width in every direction. It presents in very many places heaps and aggregations of small cells, which are perhaps more numerous around the vessels than at other portions; frequently no vessels can be seen in the section near them, and they are found placed in clefts surrounded by firm bands of fibres. The lower surface of this layer, next the muscular coat, shows a pretty even, level surface; but its upper surface, towards the mucosa, is seen thrown up in folds like the normal rugæ of the stomach. Near the upper surface the cell-aggregations are more numerous and stain more deeply than in the lower depths of its tissue.

The division between the latter layer and the mucosa is made by a broad layer of basement membrane. This basement membrane at its broadest part occupies three-fourths the width of the field of view of a one-fourth inch objective with the B eye-piece, tube out, magnifying 350 diameters. Examined under low power (50 ×), this layer appears as a band, less well stained than the subjacent submucosa, and strikingly in contrast in this respect to the comparatively brilliantly colored overlying mucosa. Under the high power the basement membrane is but poorly differentiated as to its constituents from the submucous tissue, and in places seems to pass over directly into it. It is, however, everywhere distinctly marked, either by differences of appearance or by the heaping up of small round cells just below its under boundary. The basement membrane appears at most places to be made up in the following manner. On the surface of the submucosa is a thin band of fibres running parallel to the surface, and from this

pass up fibres, which either end in spiral fibres, or, running vertically upwards, partition off the basement membrane into compartments; these compartments form two rows, bounded on two sides by the straight fibres, running parallel to the length of the basement, and on the other two sides by fibres running transversely and given off laterally from the straight fibres. These compartments generally seem to be filled with fibres derived, as already stated, from the limiting bands of the basement membrane, which within the compartments twist in spiral figures towards the centre; sometimes the fibres appear arranged concentrically. Within the compartments are seen small cells or nuclei, apparently having a direct connection or attachment to the fibres which fill these spaces.

The basement membrane, therefore, viewed as a whole, is composed of three bands of straight fibres,—an exterior, a middle, and an interior (towards the mucosa) set. Between these bands are the compartments (so called) which are divided by transverse bands.

The continuity of the basement membrane is broken at several places by clefts, through which pass tube-like structures, the nature of which it is difficult to determine with certainty. Some of them are, I think, capillaries,—this view is rendered probable from their close relation to vessels situated in the submucosa; others resemble lymphatics; and one is apparently a tubular gland. (In all the section examined, in which the appearance described is seen, these structures show only portions of their whole extent, either of their length or of their calibre, so that their exact nature must be left undetermined.)

At many parts of the mucous-membrane surface the effects of ulceration are visible, and at the other portions, which are healthy, the surface is much changed by post-mortem action. The thickness of the mucosa is evidently increased. At the deeper portions of this layer only can the structure and condition of the parts be well studied. In some portions the tubular structure is well maintained, although the cells are highly granular; at others, whilst the outlines of the tubes can be made out, the cells are seen to be greatly degenerated, and for the most part loosened from their connection with the basement membranes of the tubules, and many of the tubules are stuffed with granular débris. Many tubules also are dilated, some still maintaining their epithelial lining, whilst others, which are dilated into cysts, have as their limitary membrane only the thickened basement membrane of the tubule. The intertubular connective tissue is everywhere increased, mostly presenting a coarse-fibred condition, but in

others very delicate spindle-shaped cells are visible. In a few places the terminations of the tubules at the basement membrane of the mucosa show distinct budding dilatations, like the arrangement of a racemose gland; in one instance three bud-like projections were seen from one tubule. In general the condition of the mucous membrane is that due to a chronic catarrhal inflammation, followed by ulceration.

*Remarks.*—This case is a remarkable one from the very great alterations found in the stomach,—remarkable from the comparatively slight evidences of disease of this organ witnessed during life. From the history of the case, it is not very easy to determine with accuracy the beginning of the process which produced the morbid appearances found after death. The life-history of the case, as given by the man, is probably not a full one, as so commonly happens with patients of his class. It is clear that for a long time previous to his death he had been subject to disease of the digestive apparatus, which had at times culminated in severe diarrhœa or dysentery. His army-life, both during the war and since then, would certainly have acted as a predisposing cause. The clinical history shows, and the post-mortem examination confirms the fact, that he was not subject to several of the predisposing causes of chronic gastritis. He did not have lung or heart disease; he was not a drunkard; there was no evidence of malarial poisoning; and there was no cirrhotic condition of his liver.

There are three factors in the production of the morbid condition which remain for consideration. Three years before admission to the hospital he received a contusion of the abdomen, which was certainly of a severe character, as the history shows. There is no evidence that this injury directly affected the stomach; the position of the organ, to a greater or less degree, shields it from direct violence, except from blows aimed particularly towards its position. The patient seems to have recovered from the accident completely, and subsequently, as well as at the time, the violence does not seem to have produced gastric symptoms. The illness from which he suffered two years previous to admission, while in New Orleans, is of so uncertain a character, and the details of his condition at the time are so meagre, that no positive connection can be shown between its nature and the disease which produced death.

That the patient had had dysentery, and subsequently was affected with diarrhœa, seem to me to be the leading features in the case. Whether the dysentery and diarrhœa were symptomatic of long-existing and slowly-increasing changes in the mucous membrane of the

whole alimentary canal the history does not indicate, or whether the attack of dysentery was the initial symptom of the disease. The question of lead-poisoning being the origin of the disease can, I think, be passed by, as such a theory is entirely unsupported by evidence.

We are compelled, I think, to conclude that the morbid condition was the result of disease commencing in the mucous membrane of the alimentary canal; and it remains to be discussed whether the trouble commenced in the intestine (the colon) and was propagated upwards, or whether the beginning of the disease was in the stomach, and that from it the intestinal tract became involved also.

I am not inclined to accept the first one of these views, viz., the propagation of the inflammation upwards from the bowel to the stomach, although, as is known, an involvement of the gastric mucous membrane does result in severe chronic dysentery, not, however, without producing in the first place grave disease of the liver. The simple propagation of a catarrhal inflammation *upwards* from one extremity of the alimentary canal to the other, and the production of such alterations of the stomach as are found in this case, are unknown.

The supposition that the disease began as a catarrhal affection of the gastric mucous membrane and extended downwards, perhaps complicated with an intercurrent attack of dysentery and also by the injury received by the abdominal viscera, satisfies more completely the state of organs found at the autopsy, and is entirely consistent with the clinical history as given by the patient.

The disease commenced most probably in a subacute gastritis, and passed under the head of dyspepsia, of which the patient took little or no notice; we have no account of any acute attack, unless his illness in New Orleans be of this nature. After the changes of the gastric mucous membrane were once established, the disease must necessarily have become a progressive one, and would be aggravated by successive attacks of dysentery and diarrhœa, and would also have been strongly provocative of such attacks.

That a stomach should be so altered in its anatomical structure, and necessarily also in its functional action, is, I think, to be accounted for naturally by one of the most prominent changes in it as seen by the microscope, viz., the very great hypertrophy of its muscular fibres. Ordinarily in chronic gastritis, the distress from which patients suffer is due in a large degree to the food remaining in the stomach undigested, and, by decomposition and fermentation, produces gases and acids of a nature highly irritating to the diseased mucous membrane.

In a stomach such as found in this patient, the enormously hypertrophied muscular wall would, by contraction, rapidly hurry the food into the intestine, not only before it had digested, but before decomposition took place; hence the stomach would be relieved of the cause of the usually occurring pain and distress. But, while this condition of the muscular wall of the stomach tended to relieve that organ, the same condition would necessarily act unfortunately for the intestine. No more common source of intestinal catarrh exists, or one that acts with more energy, than the presence of undigested food in contact with the intestinal membrane; and to this condition of the stomach we must look for the explanation of the anatomical changes seen in the bowel. *February 26th*, 1880.

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9. *Specimens—liver, stomach, and duodenum—from a case of catarrhal jaundice—Enormous distension of the hepatic duct.*

Presented by Dr. Frederick P. Henry.

I. P., a fisherman, of temperate habits, æt. 63, was admitted to the Episcopal Hospital on May 15, 1880. His illness began five weeks before, and its onset was so insidious that he was unaware of anything being the matter with him until a friend remarked upon the unusual yellowness of his skin. Since then he has had occasional pains in the stomach and right shoulder. On admission, the icterus was intense, and the urine, which was acid, non-albuminous, and of specific gravity 1013, responded decidedly to the test for bile-pigment. The bowels were loose, and the stools clay-colored. The tongue was heavily coated. The abdominal walls were slightly œdematous, and fluctuation could be obtained on palpation in the lumbar regions. There was no vomiting from the date of the patient's admission, and no complaint of pain, although there was some tenderness in the epigastrium. The symptoms, from the time of admission until death, which occurred on May 23d, were gradually increasing weakness and stupor, with diarrhœa amounting usually to three stools daily. The treatment consisted chiefly in the use of nitrate of silver and opium pills and gallic acid.

At the autopsy more than a quart of serum was found in the peritoneal cavity. The tissues were all deeply stained with bile pigment, and this was particularly noticeable in the intima of the arteries. The

only organs in which anything abnormal was observed were the stomach, duodenum, biliary ducts, gall-bladder, and liver. The mucous membrane of the anterior wall of the stomach was greatly thickened and thrown into numerous prominent ridges, which, by their intersection, produced an irregularly-rectiform arrangement. On its posterior wall, and in the line of the lesser curvature, were signs of a more acute catarrhal process, consisting of redness and punctiform hemorrhages, with thickening of the membrane. In the duodenal mucous membrane the changes resembled those of the lesser curvature of the stomach, but were more marked. Near the orifice of the ductus communis was an ecchymotic patch about half an inch long. The common duct and the gall-bladder were both slightly dilated, but the most conspicuous abnormality was to be seen in the hepatic duct, which was dilated almost to the size of the normal gall-bladder. The distension caused it to lie in contact with the gall-bladder, giving rise, on first inspection, to the supposition of a supplementary bladder. This view was dispelled on observing the extreme thinness of its walls and the different character of its contents as compared with that of the gall-bladder. The point of stricture was not determined, as a probe could be easily passed along the common duct from the duodenum into both the gall-bladder and the dilated hepatic duct, and pressure upon either caused bile to flow into the duodenum. The bile in the dilated hepatic duct was of a pale-amber color, and of low specific gravity, while that in the gall-bladder was viscid and blackish-brown in color. The liver was somewhat enlarged, owing to a great engorgement of bile identical in appearance with that in the hepatic duct. Section into different parts of the gland was followed by a copious flow of bile.

The case was one of catarrhal jaundice, in which the obstruction to the outflow of bile was so great and long-continued as to cause dilatation of both the gall-bladder and the hepatic duct, moderate in the case of the former, extreme in that of the latter, where the backward pressure was more directly exerted. The distension finally resulted in complete atony of the walls of the dilated duct. *May 27th*, 1880.

### 10. *Carcinoma of the liver.*

Presented by Dr. Ferdinand H. Gross.

Mrs. Elizabeth E., widow, native of Germany, aged 60 years, was admitted into the German Hospital, March 9, 1880, and remained under the care of my colleague, Dr. J. M. Barton, until April 1, the beginning of my term of service. The early history of the case is incomplete. At the time of her admission to the hospital she complained of pain in the right hypochondriac region. On examination, a very hard tumor, about the size of an orange, could be distinctly felt at this point. According to her own statement, this had been coming on for about a year and a half. Previously she had enjoyed good health. Her appetite was usually very poor, and her bowels always remained obstinately constipated, requiring the occasional administration of strong cathartics and enemata. Otherwise, her general health at the time of her admission did not appear to be much affected. The tumor, however, soon began steadily to enlarge, and became more painful, so that manipulations about this region were resisted by the patient. Her appetite failed, and her strength rapidly declined. On the 13th of May vomiting began, which in a few days became entirely uncontrollable. Some nutritious fluids were injected into the bowels, but the patient became daily more feeble, and died on the 25th instant, retaining consciousness until the last.

The post-mortem examination revealed the hard tumor in the liver here presented. The external examination showed the hardened mass to be continuous with the liver, and to extend about four inches below the edge of the ribs and to the umbilicus. It had become firmly adherent to the adjacent portions of the abdominal walls, to the pyloric end of the stomach, and to the transverse colon. The growth is about the size of a large fist, spherical in shape, and everywhere firmly attached. No symptoms of disease of the head or chest were ever present, and these cavities were therefore not examined.

I am indebted to my resident, Dr. H. S. Bissey, for some brief notes on the clinical history of the case. *May 27th*, 1880.

*Report of the Committee on Morbid Growths:*

"A section of the new formation in the liver is found to consist of a delicate stroma of fibrous tissue arranged to form alveoli, in which are seen large epithelial cells. The tumor is an encephaloid carcinoma.

*June 24th*, 1880."

### 11. *Carcinoma of the liver.*

Presented by Dr. C. Seiler for Dr. Collins.

Thomas McLaughlin, æt. 67 years, Irish, twenty-eight years in America, was well until December, 1879, when he began to be weak, and suffered from diarrhœa; liver suddenly enlarged, and continued to do so until death. Lungs and kidneys normal. Liver filled nearly two-thirds of the abdominal cavity; would weigh about fifteen to eighteen pounds.

A microscopical examination of the growths in the liver showed them to be carcinomatous. *May 27th*, 1880.

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### 12. *Carcinoma of the pylorus.*

Presented by Dr. James Tyson.

*Specimen I.*—G. E. C., aged 32, a grocer and provision-dealer, had always been dyspeptic, but never otherwise seriously ill until February, 1879, when he is said to have had an attack of diphtheria, from the effects of which he had only partially recovered when he returned to his business. He consulted me first about the 25th of April, 1879, with a slight sore-throat, but chiefly on account of an extreme feeling of weakness. His pulse was frequent and feeble. A careful physical exploration failed to discover any affection of the heart and lungs. The usual restorative measures—iron, quinine, digitalis, good food, a visit to the sea-side—all failed to relieve him. For a short time I thought him hypochondriacal, but was soon satisfied that his illness was earnest. About the 15th of May I put him to bed. About this time, also, I was impressed by his extreme pallor. His hands and fingers appeared almost bloodless, and it was with difficulty that I secured a drop of blood from his finger for microscopical examination. The drop itself appeared like a drop of serum, and I thought the number of colorless corpuscles was slightly increased, but no careful count was made, and the observation cannot be said to have any value. Nevertheless, I concluded that I had to do with one of the set of cases of which leukæmia and pernicious anæmia are examples. My colleague, Dr. Pepper, saw him, in consultation with me,

on June 7, and we together gave him a thorough examination, which resulted in no further development, but rather in a confirmation of my original opinion. Under the use of dialyzed iron, quinine, arsenic, sulphate of strychnia, milk, and inunctions with cod-liver oil, he seemed for a time to improve. His pulse, which had been 96 to 100, and still further increased on the slightest exertion, was often as low as 84, though easily accelerated; his color seemed better; and on pricking the fingers, about June 20, the drop of blood obtained appeared brighter red in hue, although in the mean time he had had a sharp attack of diarrhœa and of hemicrania, which yielded only to large doses of quinine and hypodermic injections of sulphate of morphia. Some headache continuing, the iron was discontinued on the 28th of June, but recommenced July 7 and continued, with the arsenic, until July 24. The very liberal diet instituted was borne very well until the 19th of July, when for the first time his stomach gave way. For three days he vomited in an undigested state all food taken, and a milk diet was instituted. Repeated examinations of his stomach in the course of his illness had failed to discover a tumor, but at this time (July 21) I was able to mark out an indistinct area of dulness, about two inches in diameter, to the left of the median line, partly above but chiefly below the umbilicus. This area subsequently disappeared, to reappear again in a slightly different locality. Thus, at another time it was above the umbilicus, occupying the triangular space bounded on the right by the median line, the left by the oblique receding border of the ribs, and below by a line drawn transversely through the umbilicus. Between these two extreme situations it occupied others at different times, being generally, however, to the left of the median line until towards the close of life, when on account of its increased size it extended somewhat to the right.

The first attack of vomiting occurred, as stated, July 19. The milk diet was continued until July 27, when some solid food was permitted, at his urgent desire, and on August 1 occurred the second attack of copious vomiting. The vomiting was at night, and the undigested food of the morning was rejected. It was repeated on the 6th, 7th, and slightly on the 8th, on the 9th, and on the 13th. From this time vomiting occurred frequently, although the intervals were sometimes surprisingly long, reaching ten days, and the quantities enormous. He frequently insisted that two gallons were thrown up before the attack ceased. The matter vomited was watery, dark in color, containing sometimes food undigested, but often appeared almost like pure

water. It never contained blood. Various measures were taken to relieve,—nitrate of silver, belladonna, carbolic acid, bismuth, blisters, the latter with most effect. He was always hungry, and after vomiting would eat solid food in spite of our protestations. His decline was very slow. After November he vomited almost daily, but, notwithstanding this, he went down stairs daily until within five weeks of his death. The tumor was now very evident over a large area, with the left of the umbilicus as a centre. It pulsated with every contraction of the heart; indeed, this pulsation was evident in August.

The following extracts from my notes give an idea of his condition:

On December 2 I have, "Mr. C. keeps wonderfully well, although his condition is variable. Three days ago he was very much prostrated, having vomited nearly all day. The quantities he vomits are sometimes enormous,—more than a gallon at a time. There has been for some time œdema of the feet, and every time he vomits thus copiously the swelling goes down, as though there was a transudation from the blood into the stomach during or succeeding the vomiting."

On December 15 I have this note: "Mr. C.'s symptoms slowly grow worse; scarcely a day passes that he does not vomit large quantities. The tumor can now be distinctly felt to the left of the median line above the umbilicus, between this and the free edge of the ribs. The dulness is also extending a little to the right of the median line."

On January 12, 1880, the left leg, which, together with the right, had become very thin, began to swell, and soon acquired enormous dimensions, with pain along the course of the femoral vein. The involvement of the single leg, the pain, the degree of the swelling, forced me to the conclusion that it was of embolic origin. At this time, also, the pain in the region of the tumor became severe, and the skin over the whole epigastric and left hypochondriac regions was evidently raised by the tumor. Under the use of bandages the œdema completely disappeared except in the foot and lower leg, in which it remained, in common with that in the other leg.

On February 18, 1880, we began to administer nutritious enemata, first resorting to milk, and milk and egg together, beginning with half a pint morning and evening. We soon added, indeed almost completely substituted, the preparation of pancreas and chopped meat suggested by Professor Mayet, of Lyons, in the *Gazette Hebdomadaire* for November 21, 1879. The pancreas (one-quarter pound) was bruised alone by means of an old-fashioned potato-masher; this was then thoroughly mixed with half a pint of tepid water in which a raw

egg was beaten up ; this was then placed in a saucepan with one-half pound of finely-chopped lean meat, and placed on the back part of the range at a temperature of 100° F., where it was allowed to remain two hours. The resulting mass was then strained through a cloth, and six ounces by weight (being about one-half) injected; the remainder was injected twelve hours later. Thus an easily soluble, *partially digested* substance was injected, unaccompanied by débris of a kind which could irritate the rectum or interfere with absorption. The result, so far as absorption was concerned, was perfect. The functions soon so adjusted themselves that everything injected was retained, and a typically normal passage from the bowels obtained daily.

The nature of the malady was such, however, that no practical advantage was derived from this method of nutrition beyond, perhaps, a short prolongation of life. He grew weaker, and died March 8, 1880. For two days before death his condition was most distressing. If not actually vomiting, he was the victim of an intense and unceasing nausea, and even a short time before his death he insisted upon placing his finger in his fauces to produce vomiting.

The post-mortem examination was made March 10. The body was, of course, extremely emaciated. The abdominal cavity alone was examined. On laying back the abdominal walls, a hard, round tumor, apparently ten centimetres in diameter, was found, occupying the situation so often defined on percussion, just above the umbilicus, mostly to the left, but partially also to the right, of the median line. This, on closer examination, was found to be in the pyloric end of the stomach, projecting altogether into the cavity of the organ. The liver was adherent by its under surface to the corresponding external surface of the stomach, and the portion of the organ thus attached was removed, and is seen with the specimen, but the cancerous growths seemed to be altogether of the mucous or inner surface of the stomach. On laying open the stomach, the pylorus appeared to be occupied by a large white ball eight centimetres in diameter, but perforated in its middle by an opening sufficient to admit the little finger. The stomach itself was enormously dilated, and filled with at least a gallon of thin, slightly-colored fluid. It seemed as though the fluid would never be emptied through the very considerable opening which I made. There appeared to be no ulceration of the stomach, and, although the surface of the tumor was uneven, suggesting the external contour of a cauliflower, there was no ulceration.

*Specimens II., III.*—The second and third specimens presented, representing carcinoma of the stomach with carcinoma of the liver, the latter probably secondary to the former, were derived from J. B., a male patient aged 50, admitted to the Philadelphia Hospital, March 29, 1880. He was an Irishman, was in the army during the late war (1862), had been variously occupied as a laborer, and immediately before his illness had been working in a large boarding-house. He had been very intemperate, had had *mania a potu*, and while in the army, intermittent fever, but otherwise had enjoyed good health until his present illness. He was not aware of an instance of cancer in his family.

About March 1, 1880, he began to suffer from nausea, and a little later to vomit his food. He himself sought relief by eating less, taking but two meals a day. Burning pain and acid-bitter eructations were constant. On admission he was emaciated, feeble, sallow in appearance, and vomited constantly. He frequently caused himself to vomit at night in order to relieve the burning pain. The matter vomited never contained blood, nor did it exhibit the "coffee-grounds" appearance so often described. All food was retained for some time before it was vomited, as we learned by observations, for he himself said that he vomited immediately after eating. Milk was best retained. He was not jaundiced, had not been, and did not become so before death. There was pain upon pressure, most marked at a spot two inches below and to the left of the ensiform cartilage, but tenderness existed over the entire region of the stomach and liver. Palpation also revealed an uneven nodular surface in the portion of the epigastrium occupied by the liver, but distinct physical signs of a tumor of the pylorus were wanting. This fact, taken in connection with the man's early statements that he vomited immediately after eating (which our own experience subsequently proved to be erroneous), caused us at first to suspect that there was cancer at the cardiac orifice. Later, however, (April 27) the signs of tumor both of the stomach and liver became more distinct. The latter organ enlarged rapidly, although it did not attain the size sometimes reached; the lower border was bounded by a line extending from a point one inch above the crest of the ilium through another two inches above the umbilicus. The peculiar bossillated feel of the cancerous organ was evident through the thin walls of the abdomen. The position of the tumor of the stomach, although not fixed, was that most usual for cancer of the pylorus,—the neighborhood of the umbilicus. There was evident dilatation of the stomach,

the greater curvature of which extended almost to the crest of the left ilium.

No treatment was of service. We washed out his stomach with a siphon-tube, but it gave him no relief: indeed, he complained of faintness after the operation. As much as four quarts of water were introduced at a time with a view to measuring the degree of dilatation. Enemata of milk were used, but failed to prolong his life, and he died May 11, 1880.

The autopsy was made twelve hours after death, by my resident physician, Dr. William S. Long, to whom I am also indebted for the notes whence this history is taken. Emaciation was extreme, the abdomen scaphoid. Above the umbilicus were evident through the thin abdominal walls several slight prominences, and a ridge extending from the crest of the right ilium to the floating ribs of the other side—the lower border of the *liver*—as was confirmed on laying back the abdominal walls. The liver weighed five pounds fifteen ounces, and was a beautiful illustration of disseminated nodular cancer of that organ. Scattered over the upper and anterior surface were no less than thirty-five nodules, ranging from two millimetres to twelve centimetres in diameter. They were opaque-white, and in almost every instance typically umbilicated. It was these which could be seen and felt through the abdominal walls. The neighboring lymphatic glands were somewhat enlarged, but there were no adhesions between the liver and any other abdominal organ.

The *stomach* was only partially visible below the edge of the liver. The pylorus lay behind the liver and in the median line, and was the seat of the spherical growth exhibited, somewhat smaller, but in other respects similar to that of the first specimen. The stomach was dilated, and contained about a pint of partially-digested food of the consistence of gruel. The pyloric orifice was much constricted.

The *kidneys* were smaller than usual, but the capsule was easily stripped off. On section, the organs appeared somewhat fatty to gross examination.

The *heart* was small, but its valves were normal.

The *brain* weighed forty-one ounces. There was an area of superficial softening three centimetres across at the base of the left frontal convolution, and a similar one at the base of the left sphenoidal convolution.

*Specimen IV.*—The fourth specimen, also of carcinoma of the stomach, was taken from a German baker admitted to the Philadelphia

Hospital, December 11, 1879. His symptoms appeared about ten months before admission, and were described by himself as a "choking sensation" at the pit of the stomach when taking food, which he immediately vomited, whether liquid or solid. This condition was not, however, suddenly acquired, but gradually, and he was able to work for some time after the first symptoms appeared. He emaciated greatly, and on admission he was wan and thoroughly cachectic in appearance. A few days after admission his feet began to swell, but, on examination, his urine was found normal. This swelling subsided when he was put to bed, but immediately recurred on rising. A liquid diet was well retained, but he constantly complained of a lump in the epigastrium. Physical examination discovered no signs of a tumor, while an œsophageal bougie passed with but slight resistance at the cardiac orifice. Much relief to his distressing sensations was apparently afforded by a powder containing gr. viij of saccharated pepsin, gr. v of subnitrate of bismuth, and gr. $\frac{1}{4}$ of calomel, but he gradually failed, and died March 16, 1880, while I was temporarily off duty.

The post-mortem examination discovered the stomach now presented. The members of the Society cannot but be impressed by the contrast it bears to the other specimen exhibited this evening. The extensive ragged ulcer, with sloughing floor, extending over nearly one-half of the surface of the organ, which is itself reduced rather than increased in size, is a conspicuous feature. This reduction in the size of the stomach, accompanied by a reduction in its cavity, is ascribed to a contraction and retraction of the submucous tissue of the organ in the vicinity of the carcinomatous growth, the exact histo-genesis of which is not thoroughly understood, although Rindfleisch[1] says he feels convinced that the pre-existing tissue is really in some sort used up, and that the cirrhous growth represents the quantity of connective tissue so employed.

As to the histology of these specimens, the first two are examples of hard cancer of the pylorus, the nodules in the liver of the second case being also of the same nature. The third specimen had been so carelessly kept before it fell into my hands that it was impossible to make out its minute structure; from the macroscopic appearances, however, the specimen is probably one of soft cancer.

Dr. Henry said that the question of the disease being leukæmia,

[1] Sydenham Society's Transl. of 1872, vol. i. p. 462.

which had arisen in an early stage of the history of this case, and had been decided affirmatively, would have been decided negatively by a count of the blood-cells. The appearance of an increase in the white cells was deceptive. They might have been absolutely diminished in number and relatively increased. In a recent examination of blood which he had made, Dr. Henry had experienced the same difficulty in obtaining a drop of blood from the finger-pulp. The drop was exceedingly pale, and, when drawn into the fine capillary pipette used in the examination, resembled serum. The case was one of supposed pernicious anæmia, but had been supposed to be leukæmia by a previous observer, who had examined the blood by placing a drop of blood on a glass slide. The white cells were *relatively* increased. An examination with the hæmacytometer showed an actual diminution in the number of the white cells, and by far the smallest number of red cells Dr. Henry had ever encountered, their number per cubic millimetre being only five hundred and twenty-five thousand, the normal number being about five millions. At the time of the examination of this case Dr. Henry had under his care a case of cancer of the pylorus, in which, for purposes of comparison with the former, he counted the blood-cells. The patient was a female betwen fifty and sixty years of age. She was excessively pallid and greatly emaciated, the skin being wrinkled from loss of fat in various parts of the body, especially over the abdominal walls. Food was seldom retained for any great length of time, vomiting being of daily occurrence. Notwithstanding, the blood-cells were much more numerous than in the case of pernicious anæmia, more than three times as numerous. A count showed one million seven hundred and fifteen thousand per cubic millimetre, and also a slight diminution of the white. If examined in the ordinary coarse manner the white cells would have seemed to be increased in number in this case also. The object of these remarks is to show the impossibility of making a correct diagnosis in many cases of blood disease without the use of the hæmacytometer. *June 24th*, 1880.

13. *Carcinoma of liver, with metastasis to the brain and other organs—Destruction of the occipito-temporal convolution without affection of special sense.*

Exhibited by Dr. H. F. Formad for Dr. H. C. Wood.

T. C., æt. 42, came to the nervous wards of the Philadelphia Hospital early in September, 1880. He stated that he had been a heavy drinker, but had enjoyed good health until the last of August. On September 2 he was seized with giddiness, which lasted over twenty-four hours, but which did not leave him paralyzed. He affirmed that this was the first spell of the sort he ever had, and denied syphilis, headache, or any other known cause or premonitory symptom of the attack. Since the period of unconsciousness he has suffered from continued headache, loss of memory, giddiness, and frequent vomiting, the latter occurring without connection with the state of the stomach as to food, and not rarely in the night, as well as in the daytime.

*September 15th.*—There were no signs of local palsies, and no disorder of vision, and the gait was perfect. There was partial bilateral deafness, which he states was of long standing; there was no anæsthesia. No disorder of the taste or smell was detected, although neither was closely looked for. The right hand exerted a power of 85, the left hand of 92. The liver was found to be very much enlarged, hard, and nodular, and the diagnosis of hepatic cancer was reached. There were no changes in his nervous symptoms, and he was sent to the general medical ward, October 27.

During the last two weeks of his life his mental powers failed very distinctly, his speech became somewhat incoherent, and his memory exceedingly faulty. He died November 10.

*Autopsy.*—Brain normal, except the under central portion of the posterior lobe of the left side; springing from the membranes in this position was an irregularly ovoid tumor about an inch and a half in the greatest longitudinal and transverse diameters, and about an inch thick. The tumor was of elastic, hard consistence, of dark-red color, and had the appearance of a firm old blood-clot; it was not encapsuled. It had pressed upon and destroyed, by softening, almost the whole of the central portions of the occipito-temporal convolutions. The hippocampus was touched upon, but not altered to any extent. The softening had progressed so far as to open the lateral ventricle, the destruction not involving any of the central ganglia or the crus cerebri.

The membranes of the brain were abnormally thickened everywhere, and the fissures were bound together by inflammatory exudations. The one corner of the tumor had produced a small superficial spot of softening in the cerebellum; the attachments of the tumor to the surrounding tissues were very loose. A decided increase of cerebral fluid, turbid in appearance, was observed. Skull normal.

Examination of the thoracic cavity revealed pericardium and right lung and pleural sac normal. In left pleural cavity extensive adhesions and two pints of a turbid effusion were found; left lung strongly congested, and a wedge-shaped cavity of evidently embolic origin was seen.

Mediastinal and bronchial glands enlarged; heart normal. The abdominal cavity contained over one gallon of a straw-colored liquid; no peritonitis.

Liver very much enlarged; weight nine and a half pounds, of dark-brown color, and studded with the protruding, umbilicated, white nodes peculiar to cancer, varying in size from one-half inch to five inches in diameter. The head of the pancreas was also involved by similar nodes. Mesenteric glands greatly enlarged and noded. Kidneys invaded by a number of small nodules, scattered throughout the capsules as well as the parenchyma of the organs.

Stomach, intestines, rectum, and rest of organs normal.

*Microscopic examination* by Dr. H. F. Formad determined the structure of the new growth of the liver, of the brain, and of the other organs to be that of a *cylindrical* epithelioma, the primary growth having developed in the liver.

Dr. Formad added that the new growth, of the brain especially, showed typically the peculiar cones built up of cylindrical epithelium piercing through a vascular connective tissue in every direction; that at the same time, however, it demonstrated in places well the direct transformation of a cylindrical epithelioma into a simple glandular carcinoma, as described first by Perls, the solid cones of the epithelioma breaking up; and the cylindrical epithelial cells are distinctly seen *transforming* into spheroidal cells, which fill, loosely packed, the alveolar spaces. *December 9th*, 1880.

14. *Cirrhosis of the liver and kidneys—Liver deformed—Death from softening of the brain.*

Presented by Dr. J. H. Musser.

I visited the patient from whom the specimens before you were taken the first time on the 8th of October, 1879. Her condition at that time and the subsequent course of the disease are embodied in the following notes :—

Mrs. H., æt. 57, born in Ireland, a resident of Philadelphia, for the past two years living in an intensely malarial district near the Schuylkill. Different members of the family have had malaria the past three months. Married. Eleven children ; six living and healthy ; the others died of acute disease in early childhood or youth. Circumstances and comforts of life quite moderate. Considerable mental worry from the cares of a large family and on account of husband's intemperance. Habits good.

Father died of "jaundice." Cause of mother's death unknown. A sister died of consumption. No further evidence of hereditary disease.

Always healthy, save an attack of ague twenty years ago, and, for several years, frequent attacks of "biliousness." These attacks were characterized by anorexia, nausea, and vomiting of a yellow-and-green matter, with or without diarrhœa. No syphilis.

For six months patient has suffered from poor appetite, bad taste in the mouth, flatulency and acidity, and irregular bowels. The past three weeks has been quite miserable, being without appetite and very weak. Cramp-like pains, coldness and heaviness of the lower limbs have troubled her for a week, while for two days she has been vomiting.

At present (October 8) she is quite weak, somewhat emaciated ; extremities cold ; skin of trunk warm and harsh, of a dark, muddy color ; face of same color, with injected capillaries of the cheek ; conjunctiva yellowish ; heart normal ; pulse 130, weak ; lungs normal ; breathing hurried, thirty to the minute. The hurried pulse and respirations are due to the exhaustion from vomiting. Nausea is constant ; vomiting occurs every half-hour. The matter vomited is a yellowish, sour liquid, with flakes of mucus in it ; no blood. Soreness over stomach, but no pain.

Inspection of abdomen reveals a slight general enlargement, with localized bulging in the right hypochondrium. No enlarged veins.

On palpation, there is found a smooth, hard tumor, extending from the outer border of the right rectus muscle to the axillary line. The lower margin is, at the outer border of the right rectus, one inch above a horizontal line through the umbilicus from left to right. In the nipple-line it extends to the line, in the axillary region to an inch above the crest of the ilium. No pain attends its handling. The upper boundary of the liver begins at the fifth rib on deep, in the sixth interspace on superficial, percussion. From thence dulness extends to the margin defined by palpation, being continuous with the tumor-dulness. The left hepatic lobe is not enlarged. Normal liver-dulness not increased posteriorly. With inspiration and expiration the entire mass moves, so that it is, with the evidence of percussion, connected with the liver. Spleen is not enlarged. Bowels are costive.

Appropriate diet and sedatives soon allayed the gastric symptoms, and in three days she was ordered cinchona, strychnia, and muriatic acid, and a regulated diet.

About three weeks afterwards her urine burned her very much when passed. It was found to be of an acid reaction, a dark-red color, without albumen or sugar, containing urates and bile-pigment.

In November she had a sudden, profuse, painless uterine hemorrhage. Her menses had ceased years ago. This was the only hemorrhage throughout her illness.

I saw her again December 11. She had emaciated somewhat. She was very weak. Her eyes were jaundiced, the capillaries of the face congested, and the skin of the face dark or bronzed, as previously noted. She had some slight fever, and a pulse of 110. Her appetite was poor, tongue coated brownish, red and glazed at the edges, thirst great, bowels regular. Would not allow an abdominal examination. Suffers from pain in the loins. Urine is scanty. A dry, hacking cough annoys her very much. At times a dark-yellow expectoration. No physical signs of bronchitis. Slight œdema of the feet.

12*th*. Œdema subsided somewhat. Nausea and vomiting all night. Vomited matter at first like the yolk of an egg, later a watery fluid with flakes of green-tinged mucus floating in it, and green masses at the bottom of the vessel.

13*th*. Vomiting better. No fever. Pulse 100. Liver as before noted; descends three inches, and is pushed to left by ilium. No œdema. Colic and diarrhœa last night. Much stronger. Urged continuance of the previously ordered acid tonic.

23*d*. Another gastric attack, as previously.

*January* 16, 1880. Noted as follows: Been visiting her every week, and find mind gradually failing. Wanders readily from a subject, talks of unreal occurrences, and of nonsensical matters. No memory. Sleeps poorly. Slight headache. Urine scanty, very red; specific gravity 1020; slight albumen. No jaundice. Considerable tympanitis.

21*st*. Urine high-colored, acid; slight amount of albumen; no sugar; bile-salts and pigment. Microscopically, bladder-epithelium, pus, uric acid, and an abundance of phosphates. Growing weaker and weaker. Dry, brown tongue; slight gastric pain; no vomiting; regular bowels; slight ascites; no headache. Poor sleep; wandering delirium; refers to imaginary occurrences. Is continually enacting the movement of winding a spool of thread.

31*st*. Peritoneal fluid increased. Pain in abdomen. Temperature not elevated. Pulse 120. "Some smothering about the heart." Mind less composed than before. Urine scanty.

*February* 2, 1880. Urine scanty; eight ounces passed in twenty-four hours; high-colored. Microscopically, red blood cells in abundance, a few white cells, epithelium from kidneys, ureters, bladder, and vagina; a few crystals of oxalate of lime. Urates, phosphates, and tube-casts absent; bile-pigment in abundance. Chemically, bile, one-sixteenth albumen; no sugar.

5*th*. Ascites increased. Tongue flabby, red, and fissured. Vomited once—bile. Bowels regular, feces dark-colored. Pulse 120. No œdema of feet. Feeble; more stupid. Recognizes with difficulty, and comprehends poorly. At apex of heart a "murmurish" sound.

*March* 2. Vomiting a dirty-yellowish, offensive, copious matter. Diarrhœa past two days; stools thin, clayey, offensive. Urine passed involuntarily. Is semi-conscious; aroused with difficulty, and murmurs unintelligibly. Lies on right side, with the knees and legs drawn up and head bent to the chest; lack of co-ordination in movements for a week. Past three weeks face has had the appearance of dementia, as in cerebral softening; not the cirrhotic countenance.

12*th*. Pulse 200, moderately strong, regular; respiration 32, short. Twitching of the muscles of limbs. Can be aroused. Some hyperæsthesia; slight fever. Does not take food; urine and feces passed involuntarily.

Became more and more unconscious, and died on the 13th in coma.

*Autopsy.*—Examined twenty-four hours after death, Dr. W. E. Hughes assisting.

Rigor mortis not well marked; body not extremely emaciated; skin dark, dirty color; no anasarca or jaundice.

Abdominal walls one and a half inches thick; fat moderately abundant. Cavity of abdomen almost filled with a clear, light, straw-colored fluid; no flakes of lymph, and no adhesions; no congestion of the visceral or parietal peritoneum. Liver not displaced, occupied the position defined during life; other organs in normal position. The liver was not nodular, but its surface was granular, and on section it was dense and firm, granular with increase of connective tissue. A slight indentation marked this. The right lobe measured five inches from right to left, the left three inches. From above downwards the right lobe measured seven and a half inches, the left three inches. The right lobe was two and a quarter inches thick, the left two inches. It weighed two pounds two and a half ounces. Spleen normal in size, dense. Kidneys enlarged, congested, weighed ten ounces each; capsule easily removed; proportion between cortical and medullary portion normal. Mucous membrane of stomach and intestines pale.

Heart, left ventricle contracted; mitral valve slightly thickened. Lungs slightly œdematous. Brain not examined.

Fluid of peritoneum contained albumen, sugar, and urea. Kidneys, microscopically, were congested, and in early stages of cirrhosis. Liver markedly cirrhotic.

*Remarks.*—The cause of the enlargement only of the right lobe of the liver, and only of the anterior portion, at first puzzled me. It could not be a hydatid, or a purulent or serous collection between the liver and diaphragm pushing the organ down, for the lungs were not encroached upon, nor were there any other confirming symptoms of such conditions, as fluctuation or fever. There were no symptoms or signs of disease of surrounding organs, as the kidneys, to cause a dislocation of the liver. Hydatids, abscess, and cancer were excluded, from absence of symptoms peculiar to each, although the indefinite cause of the father's death made me consider cancer carefully. The knowledge of the patient's habit of tippling, which I learned after her death, would have confirmed my suspicion of cirrhosis. Before the symptoms of the second stage of cirrhosis developed it was only suspected; for in the first stage the liver is enlarged uniformly; this was of one lobe. The symptoms of the second stage confirmed my suspicion, although there was no contraction.

The commencing Bright's disease was aggravated by the ascites, no doubt. The amount of fluid was never so great as to interfere with respiration or cardiac action. The state of the mind forbade tapping.

The cause of death was undoubtedly softening of the brain. The renal complication was not sufficiently grave to cause uræmia. The cirrhotic cachexia was not sufficiently developed to produce the cerebral symptoms of the later stage of cirrhosis.

*December 9th*, 1880.

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### 15. *Carcinoma of stomach.*

Presented by Dr. J. H. Musser.

The patient whose stomach I present for your examination applied to me for treatment on the 11th of March of the present year. From him I gleaned the following history:—

H. O., æt. 55; butcher; moderate drinker, excessive smoker; has been a hard worker; unfortunate in business, and, of late years, in poor circumstances; has had considerable mental strain; always healthy until present illness; no venereal disease; family history good; wife died of cancer of the stomach eight months ago.

Present illness was of six months' duration. Increasing languor, debility, and emaciation, dyspnœa on exertion, continual yawning, sleeplessness, and loss of memory, have been noticeable symptoms. What most distressed him, however, was a weight and fulness after meals in the stomach, with flatulence, lasting two or three hours, and only being relieved by forced vomiting; thirst has been very great; constipation persistent and increasing.

His condition when examined was as follows: Large, heavily framed; very much emaciated, having lost seventy pounds; countenance did not indicate suffering from pain, but from mental torture; face emaciated; eyes sunken; conjunctiva pale; sclerotic dead-white; lips pale; complexion dirty-yellow; extremities cold; voice weak; speech slow; sighs considerably; mind is sluggish; memory poor; heart and lungs normal; pulse 90, feeble; tongue large, pale, flabby, heavily coated; teeth poor; throat dry, slight pharyngitis; appetite fair; thirst great; after eating, weight and fulness in the stomach; great flatulence; never vomits involuntarily; when he provokes

vomiting the matters ejected are the contents of the stomach and greenish fluid; never blood; bowels very costive.

*Inspection* of abdomen reveals a pulsation between the xiphoid cartilage and the umbilicus, and a distinct prominence to the right of the median line above the umbilicus. *Palpation* defines the bulging to be two and a half inches above the umbilicus, a little to the right of the median line, and extending within one-half inch of the ribs. *Percussion* does not define any resonance between the normal dulness of the liver's margin and the mass. No pain on pressure, and no increase of local heat; no murmur on auscultation. Liver and spleen normal size; urine not examined.

*May* 1. Learned from one of his family that he had emaciated very much, and become much debilitated.

24*th.* Called to him again, and found him extremely emaciated. Abdomen very scaphoid. Pulsation very visible. Mass observed two inches above umbilicus, between the median line and the ribs. Percussion defines the tumor in that area, extending three inches along the ribs. The size of the mass is greater than when first examined, or the scaphoid state of the abdomen causes it to be more readily outlined. No tenderness on pressure. Paroxysmal pain of a burning or sharp, shooting character. Almost constant vomiting. Matters vomited are the food and an acid, white liquid, burning the fauces and mouth. At times, however, free from vomiting, so that to relieve the distressing sense of fulness he has to provoke it. Tongue large, pale, flabby, coated; pulse 96; bowels constipated. Skin itchy, dry, with a general pustular eruption on it. No sleep; very restless.

26*th.* A powder, given every three hours, containing morph. sulph. gr. $\frac{1}{12}$, sodæ bicarb., lactopeptine, āā gr. ij, pulv. aromat., gr. $\frac{1}{12}$, prescribed on the 24th, gave relief to the pain, stopped the vomiting, caused sleep, and permitted the more liberal use of milk.

The symptoms were all better, though he took scarcely any nourishment, and on the 31st of the month he died of exhaustion.

*Post-mortem* (twenty-four hours after death).—Body extremely emaciated; abdomen concave; skin of same color as before death, very rough; rigor mortis well marked.

Abdominal cavity alone examined. Tissues bloodless on section; organs in normal position; no ascites.

*Stomach* very much dilated; vessels congested, both arteries and veins, especially near the pylorus. It contained a dark gray liquid; no blood. At the cardiac end of the stomach the mucous membrane

was very pale, becoming more congested towards the pylorus, with swelling and softening of the mucous membrane and some thickening of the walls towards the pylorus. At the pylorus the walls were much thickened, from one-half to three fourths of an inch, and projected into the stomach as a shelf. The walls of the duodenum were involved to the extent of two and a half inches from the pylorus. They were infiltrated by a dense, firm mass of tissue, softer an inch from the pylorus than at the pylorus. At some points the thickening was greater, giving the appearance of nodules. Neither the pyloric orifice nor the intestine was occluded, both admitting one finger. I neglected to state that the congested portion of the mucous membrane was bathed with mucus. The mesenteric glands were enlarged.

The *liver* was slightly fatty; weighed two pounds fifteen ounces. The other abdominal organs were natural to a person dying of chronic disease. The pancreas was normal, situated back of the mass, and removed with it.

*Remarks.*—At the time of the first examination cancer was diagnosed and an unfavorable prognosis given. I was not called in again until shortly before death; hence the absence of notes in the interval. The symptoms and course were characteristic of cancer of the stomach, and the autopsy confirmed the opinion. The microscopical appearances of the tumor are those of a scirrhus, with a few spots of degeneration. *Dec. 9th*, 1880.

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16. *Report of a case of typhoid fever (?) in a child æt. ten months.*

By Dr. Longstreth.

Dr. Longstreth said that he had been called upon to make the autopsy in this case, and regretted that the history furnished in connection with the specimens was necessarily so imperfect. He was induced to speak of the case as one of typhoid fever solely from the post-mortem evidences. The clinical history, furnished by the attending physician, Dr. Scott, was as follows: The child, female, light mulatto, æt. 10 months, nursed at the breast, taking no other nourishment, but was in the habit of drinking ordinary hydrant-water. She had been strong and well until the present symptoms set in. For two or three days previous to January 6, 1881, the mother had noticed that the child did not play or take the usual notice of things, and was

weak, but did not regard her as very sick. On examination, the child was found pale, dull, and could be roused only by extra efforts, and immediately fell asleep again. There was no desire for food, tongue coated white, bowels obstinately constipated, requiring repeated doses of castor oil, and finally an enema, to produce their evacuation. The pulse was about normal in rate, but weak. The skin was dry, but did not give a sensation of heat to the hand. There had been no epistaxis, and the lungs were free from any abnormal physical signs. During the second week the physical signs remained as noted, except that now pain in the abdomen and diarrhœa occurred. The fecal discharges were of grass-green color. Appropriate remedies relieved the pain, and the diarrhœa was easily and promptly checked. The child progressively became weaker, and remained in the same somnolent condition. She was roused and proper and abundant nourishment was taken during the whole course of the illness. Death occurred in the early part of the third week from the commencement of the attack.

*Post-mortem Examination*, made twenty hours after death.—Rigor mortis marked. Posterior parts of the body discolored. Emaciation marked. The great cavities and their serous lining showed no abnormal conditions.

The right side of the heart was relaxed, and contained considerable fluid blood and soft clots extending into the great vessels connected with the auricle and ventricle. The left side was nearly empty, the left ventricle firmly contracted. The heart-muscle was pale, rather friable, but not fatty. The lungs showed considerable posterior congestion and a few small areas of collapse, but otherwise were normal. The bronchial tubes contained considerable frothy mucus, but the mucous membrane gave no evidence of any inflammatory change, though parts of the respiratory tube were of a reddish color.

The spleen was firm, a little swollen, pale externally and internally, of a dark-reddish hue; its pulp was moderately resistant, and did not exhibit the appearance so constantly seen in the typhoid fever spleen.

The kidneys were nearly typically normal in appearance; the only alteration was a very slight swelling, and an opacity of the cortical portion difficult to differentiate from the general paleness of all the organs.

The liver was pale, its edges very slightly rounded; on section, its tissue was anæmic and appeared homogeneous. The bile in the gall-bladder, moderate in amount, was pale green and mucus-like.

The stomach exhibited a slight swelling of its mucous membrane, and contained, floating in a small amount of fluid, some milk-curds.

The intestine, throughout its extent, exhibited marked alterations. Externally, while generally pale and slightly opaque, at parts reddish patches of coarse injection were seen; but the peritoneum was unchanged.

Its mucous membrane throughout, but more markedly in some parts than in others, showed a distinct but not severe catarrhal inflammatory condition. All the Peyer's patches were more distinct than normal, and, as the examination of them progressively approached the ileo-cæcal valve, they were found more elevated and prominent. The few lower patches were very marked in this state, and their surfaces were uneven, but to the naked eye there was no evident ulcerative destruction of the mucous membrane overlying them. The patches were of unusual length, considered proportionally to the relative size of the intestine. The solitary glands of the ileum, as well as of the colon, were enlarged and prominent, and the mucous membrane of the latter also showed catarrhal changes, but to a less degree than in the small intestine. The appendix vermiformis was swollen, thickened, and gave a feeling of firmness to the finger, but contained only fluid contents.

The mesenteric glands were large and swollen, especially the ileo-cæcal gland.

The contents of the gut throughout were pale yellowish and to a great extent fluid, but some firmer masses of a yellow color were present.

The bladder was empty. The generative organs were normal.

*Microscopic Examination.*—Portions of the intestine were placed in Müller's fluid over-night, to prevent alteration until sections could be made the following morning by freezing. The Peyer's patches at the time the sections were made showed a much greater prominence than when removed from the body. This change was apparently due to a reduction in the swelling of the surrounding mucous membrane, etc., from the escape of fluid.

The lymph-follicles, as now seen with the microscope on the table, are crowded with the small cell-elements, which at the peripheral portions are arranged in straight bands, while in the central parts the corpuscular elements are heaped together in a confused manner. In some of the follicles their centres are evidently undergoing softening, or at least the fibrillar tissue is less resistant and the unhardened tissue has given way. The prominence or projection of the patch is very

great, and, as viewed with a lens of low power, is seen to exceed considerably the thickness of the surrounding intestinal wall. In one of the sections the mucous membrane overlying the follicle is seen to be removed, and the infiltrated follicular tissue is exposed, giving the appearance of a commencing ulcerative destruction.

From this brief note of the microscopic appearances presented by this specimen, it is evident that the conditions resemble those found in unquestionable cases of typhoid fever at certain stages of this disease.

*Remarks.*—As already remarked, in presenting these specimens to the Society, the clinical history is entirely defective in furnishing a proof, or even a hint, that this case was one of typhoid fever, and it is not claimed that the post-mortem evidence is decisive. However, since the knowledge of the symptoms of this disease occurring at this period of life is very meagre, and the autopsy records of the anatomical changes in the organs are so very few, we are possessed of very insufficient guides to lead us to the determination of the nature of the alterations here found.

If the case during life had been attended with prolonged diarrhœa, the presence of this symptom might furnish us with the explanation of the enlargement of the lymph-follicles and the mesenteric glands. The rapid emaciation and the condition of the other organs besides the intestines are not inconsistent with the occurrence of a febrile disease, the presence of which, owing to the absence of a thermometric record, may have been overlooked. The reasons for suggesting the typhoid nature of the disease have already been given.

Dr. Tyson thought the specimen, if not one of typhoid fever, was an anomalous one, in that the agminated glands were enlarged, although in a case of such short duration ; for in *chronic* intestinal troubles such a condition of Peyer's patches, as well as of the solitary glands, is not unusual. He would ask Dr. Longstreth whether the surrounding hygienic conditions were such as to suggest any source of infection.

Dr. Longstreth replied that the child was at the breast, but of course must have occasionally had water to drink, which might have been the vehicle of infection if such existed.

Dr. Eskridge said that typhoid fever in the young ran a different course from the disease in the adult. According to his experience, it usually ran its course in two weeks or less. In one patient under his care, where death resulted from hemorrhage from the bowels during convalescence, the fever had run its course in eleven days. Text-books did not sufficiently emphasize the difference of the symptoms and

duration of the fever occurring at the two periods of life mentioned, although within the last two years Dr. Jacobi, of New York, in one of the leading medical journals of his city, had called especial attention to the frequency of typhoid fever in children. He stated that it is milder, that it runs its course more rapidly, that the temperature rises at the beginning and falls at the close more abruptly, that the fever takes on more of the type of remittent (for which it is often mistaken), when it occurs in children. The doctor of late had been much interested in collecting cases of typhoid fever in the adult with irregular temperature records, and related cases to illustrate.

Dr. O'Hara asked whether the other children who had died in this family had shown the same symptoms, at what intervals had the fatal cases occurred, and whether anything like a constant cause could be detected.

Dr. Formad inquired as to the possibility of syphilis.

Dr. Longstreth, in reply to the last two speakers, said that, as he had merely made the post-mortem examination, and was unacquainted with the family, he was unable to say anything positively, but as far as syphilis went, the history and absence both of skin eruptions and signs of chronic wasting would indicate its probable absence.

Dr. Formad said, in explanation of his question, that the microscopic examination suggests a cirrhotic induration rather than ulceration; in fact, portions look as if the follicles were hypertrophied. Birsch-Hirschfeldt has stated that in congenital syphilis there is this increase of the connective-tissue elements in the lymphatic structures. The present specimen would seem to indicate such a condition, while the characteristic medullary infiltration of typhoid fever is absent.

Dr. Nancrede related a case of a form of fever such as Dr. Eskridge had described, of which he had seen one case, and which he had heard from his colleague, Dr. M. J. Lewis, had been quite prevalent last summer. To him, such cases seemed merely what the Germans have described as abortive typhoid. As to the present specimen, he would like to ask if any member present knew the *normal anatomical* appearances presented by the small intestine of a child of eighteen months. *He* certainly did not, and before we discussed pathological alterations it would be well to have some knowledge of the normal conditions of the parts. It is certainly true that as age advances it becomes more and more difficult to demonstrate Peyer's patches in a state of health. Dr. Longstreth has said that these patches were not so prominent at the post-mortem as now, as the mucous membrane surrounding them was

swollen and infiltrated. Their present prominence is due to the effused products having escaped while the tissues have become condensed by the reagent. May it not only be possible, but probable, that these glands are normally as prominent as in the present specimen in children of this age? *A priori*, as we know that they gradually disappear normally with advancing years, it is but fair to infer that, like the thymus and other glands, their appearance a short time after birth is far different from that to be found at but a comparatively sliglitly later period of life.

Dr. O'Hara asked whether Peyer's patches could not become enlarged from any other cause than typhoid fever.

Dr. Tyson said that experience thus far had shown no other cause to be efficient, although there is no reason why there might not be some other. In the present case the diarrhœa and acuteness of the attack precluded the idea of syphilis. As to the duration of typhoid fever in children, he thought that all authors agreed that it ran a much shorter course in children than in adults, rarely lasting in the former more than two weeks.

Dr. Longstreth asked Dr. Tyson if he was aware of any case of typhoid fever on record in an infant under one year of age.

Dr. Tyson replied that he was unaware of any such.

*Feb. 24th*, 1881.

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### 17. *Carcinoma of stomach and liver.*

Presented by Dr. E. T. Bruen.

These specimens exhibit an encephaloid cancerous growth, embracing the entire anterior wall of the stomach. The largest masses of the new formation are distributed at the junction of the œsophagus with the stomach, although the œsophagus is not involved. The mucous membrane of the stomach is not ulcerated, but the masses are very large, and as felt through the anterior wall they conveyed the idea of a triple layer of figs,—that is, a well-flattened yet decidedly thick and bulky mass, fully four inches in breadth by seven vertically. The growth has caused considerable dilatation of the stomach, especially the cardiac extremity. Extending from the lesser curvature of the stomach can be found a chain of enlarged lymphatic glands running up under the fissure of the liver, between the right and left lobes.

This chain of glands commences around the pancreas, but the structure of this gland is not involved.

Cancerous nodules are to be seen in the liver; the largest nodule, in the left lobe. In the pleura of the right side nodules are found which microscopic examination has shown to be cancerous.

I also exhibit a portion of the skull, with a developed nodule in the periosteum, of the size of a walnut.

These specimens were removed from the body of a man 40 years of age, with a good family record, without history of cancer or scrofula.

The sequence of the cancerous development is as follows: In June last, about five months prior to the date of his death, the tumor of the stomach developed. Six weeks before death, the tumor in the liver was diagnosticated; some three weeks before his death, the nodules in the pleura were discovered.

The clinical history is a recital of the usual symptoms of similar conditions. It is interesting to note that vomiting was never a symptom unless very inappropriate food was used; there was never vomiting of blood, but large amounts of gas were frequently passed per mouth and anus. There was no constipation. To explain this absence of frequently-expected symptoms, I would note that the pylorus was not involved; thus vomiting was not induced, because the contents of the stomach could readily pass into the intestine; and, as the liver was only involved in the later history of the case, intestinal digestion was not impaired.

Pain, however, was a prominent symptom, and the suffering was much increased by the developments in the pleura.

Another interesting symptom was a total paraplegia. The power of motion in the legs was abolished, and there was no control over the bladder or rectum. This paralysis appeared some four weeks before death. It fully developed itself in about one week, leading me to suspect that a similar change would be found in the membranes of the cord to that noticed in the pleura. At the autopsy the cord was examined in its entirety, and nothing abnormal could be detected. In fact, the cord has been cut into sections, and is used to demonstrate the normal histology of that tissue to the class at the University of Pennsylvania.

A loud systolic bruit could be heard over the region of the aorta, audible just below the ensiform cartilage anteriorly, diminishing as the aorta passed downward. It was heard posteriorly over the dorsal spine quite distinctly. This bruit was not affected when the hand-

and-knee position was assumed, or when an effort was made to draw the tumor downward into one or other iliac region, which could be partially accomplished. The persistence of murmur was probably owing to the constant pressure of this large mass on the aorta; and, owing to the fact that most of the growth affects the cardiac end of the stomach, it could not be dragged away from the vessel as one could drag a tumor in some other part of the stomach.

Yet, for all this, I felt I could exclude aneurism even in the early stages of the growth, since aneurismal pressure-signs were absent, and there were no evidences of systemic conditions which predispose to disease of the arterial coats, such as syphilis, alcoholism, rheumatism, and the like; and these evidences I regard as of the utmost importance.

Finally, I would say that the movable character of the tumor above referred to, and the absence of symptoms of intestinal indigestion, were important evidences that the location of the primary tumor was in the stomach. I think you will agree that cancers of the anterior wall, since they grow backward and inward, are not necessarily more prominent to palpation during life than are tumors of the posterior wall of the stomach. This tumor seemed to be situated just beneath the skin, so superficial did it seem during life. But I have examined cases in which the tumors seemed equally superficial during life, yet autopsy revealed the location of the disease in the posterior wall.

*February 24th*, 1881.

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### 18. *Primary carcinoma of the gall-bladder.*

Presented by Dr. J. H. Musser.

Mrs. McM., the patient from whom the specimens before you were removed, was under my care from the 9th of March until the 4th of May, 1881. Her illness dated from the middle of December, 1880, and continued until the 18th of May, 1881. She died in the Presbyterian Hospital, and I am indebted to Dr. Markoe for the privilege of this exhibition. The history previous to my attendance was apparently that of an intermittent fever with hepatic and gastric complications. The symptoms referable to the liver were paroxysmal lancinating pains in that region, at times so severe as to almost cause collapse. The gastric symptoms were epigastric pain, loss of appetite, flatulence, acid

eructations, and, at variable intervals, attacks of vomiting. She had not been jaundiced.

I found her suffering from an intermitting fever with daily paroxysms, each beginning about 1 P. M. and continuing until 7 or 8 P. M. The chill lasted a half-hour, and was moderately severe; the fever lasted four hours, at its height the temperature being 103°. She was emaciated; her features indicated exhaustion and were somewhat pinched; her complexion was sallow; she was anæmic.

She suffered from severe paroxysmal pains in the epigastric and hypochondriac regions. The areas were tender on pressure, but no tumor was noticed. The liver-dulness extended three inches below the ribs in the nipple-line. The spleen was twice its natural size. The appetite was lost. She had nausea, acidity, and flatulence. The bowels were constipated. Urine contained neither albumen nor sugar. Urates and uric acid were in abundance.

During the course of her illness the symptoms may be detailed under three headings,—febrile, gastric, and hepatic. There seemed to be no relation between the different sets of symptoms, except the febrile and hepatic. The fever was more continuous when the jaundice was deepest. At the risk of some repetition, I will note them.

*Gastric.*—Tongue covered with a dark-yellow, heavy fur, moist, tremulous; later, dry and brown. Bitter taste in the mouth, secretions viscid, loss of appetite, thirst, acidity, flatulence, weight and fulness after meals, epigastric pain and tenderness, nausea and vomiting. I desire to call particular attention to the nausea and vomiting. It, of all the gastric symptoms, was especially independent of any other symptoms. It occurred once in two weeks, once a week, or twice a week. Each paroxysm lasted from two to four days. The fluid vomited varied, from a clear-white, sour, to a greenish-yellow, bitter liquid.

*Hepatic.*—Pain in the right hypochondriac region, paroxysmal, sharp, and lancinating, radiating to right and left shoulders and to epigastrium; tenderness on deep palpation, absence of tumor or irregularity of edges, enlargement as noted above. On account of liver-derangement, marked jaundice, with clay-colored stools and bile in the urine. The jaundice occurred four times while the patient was under my care. The first attack was light, lasting three days; the second and third each lasted a week; the fourth, beginning on the 25th of April, continued till death. With the second and third attacks the fever was remittent in character; with the last it was continuous. As

the jaundice improved, the fever changed from the remittent to the intermittent type.

*Febrile.*—As may be inferred from the previous notes, the fever was irregular. At first it was intermitting, then remitting, and, finally, it became continued. At no time was the temperature higher than 104°. With the rise in temperature there was a corresponding pulse-rise. The increased pulse was not as great as a pulse at the height of a paroxysm of intermittent by twenty beats. The last month of illness it became rapid, feeble, compressible, and dicrotic. When the fever was remittent or continuous, in the evening there was delirium. The "typhoid" state developed the last ten days of her life, and death took place from cardiac failure. The use of antiperiodics prevented the paroxysms at first. As soon as the drug was suspended they recurred. Finally, in spite of my remedies, the fever continued.

The following facts also bear upon the case. The patient was a married woman, who had not lived with her husband for several years, on account of his brutality. She was 37 years old, a nurse by occupation. She had always been in poor health, and had had several attacks of biliary colic. The family history was good, save that a maternal aunt died of cancer. Her mother and brothers were living; her father had been killed.

The post-mortem examination was made by Dr. Warnock. He reported that rigor mortis had set in early, that the body was greatly emaciated, and that all the tissues were stained with bile. With the exception of the liver and spleen, the organs were normal for a person who had died of heart-failure. The liver and spleen I examined carefully. The latter organ was about twice the normal size, of a dark greenish-brown color, and of about normal consistence. Unfortunately, it was not examined microscopically.

The liver was of a peculiar shape. The right lobe was eight inches long, the left the same. Curiously, however, the lower edge of the latter lobe did not extend as low down as that of the right lobe, but its upper edge was three and one-half inches beyond the same edge of the right lobe, while the lobe was doubled on itself to conform with the concavity of the diaphragm. Transversely the right lobe measured five and one-quarter inches, the left three and one-half. The former was two and one-half inches thick, the latter one and one-half. The gall-bladder was in the normal position and of the normal size. It was adherent to the duodenum. The color of the outer portion was pure white, not bile-stained. It was not collapsed, and was of firm

consistence. A portion of the bladder underneath the liver, not quite as large as a walnut, was of natural color and fluctuated.

On section of the gall-bladder it was found that the white appearance of the exterior was due to cancer, which involved three-quarters of the organ. The growth encroached upon the cavity so that the walls were from one-half inch to one inch thick. The mass had the appearance of a soft cancer; the inner surface had a ragged, ulcerated appearance, and in the centre of the mass was found a gall-stone the size of a filbert, with a rough surface, looking like a mulberry calculus. The stone was lying against the mouth of the gall-bladder; behind the stone was a cavity about the size of the stone, filled with a thick greenish-yellow fluid. From the appearances one would infer that the stone acted as a ball-valve to the duct. The duct was involved to and beyond the junction with the hepatic duct. The hepatic duct was enlarged, its walls thickened, its calibre much increased. The branches extending into the upper half of the right lobe were greatly dilated, even almost to the periphery of the liver, terminating in saccular dilatations. The ducts contained a thick grayish-green matter; the walls were of a slate-gray color, dotted with dark points. The liver-substance traversed by these ducts was dark and soft, not unlike gangrene. A part in the centre about three inches square was especially of this appearance. The remainder of the structure of the organ was slightly stained with bile and fatty in appearance.

On microscopic examination the malignant mass was found to be of the nature of a medullary cancer. The cystic and part of the hepatic duct was involved. The transition from the cancerous to the catarrhal process in the ducts was well marked. In the liver, catarrhal inflammation of the ducts was very distinct. In the lymphatic spaces around the ducts there was an abundant infiltration of epithelioid or indifferent cells. It was impossible to say, though it was highly probable, that the disease had involved the ducts. No doubt such would have been the case had the patient lived longer. The cancer of the gall-bladder was no doubt primary, from the ragged appearance of the mass and the absence of nodules. I shall try to determine why the special ducts were involved, and will report the results to the Society. Dr. Formad very kindly confirmed the examination.

I desire to discuss elsewhere, in detail, the etiology, the clinical history, and the diagnosis. Remark, however, the relation of the gall-stone to the new growth as cause and effect in a person predisposed to cancer. Let me say, too, that I did not diagnose cancer of the

gall-bladder. Long before her death I suspected internal malignant disease.

Dr. Formad said that he had examined the specimens microscopically, and had readily detected the presence of carcinoma of the soft type. The gall-ducts were strongly catarrhal, and the surrounding lymphatics were crowded with a small-celled infiltrate, which might possibly be metastatic, as some of the cells had a suggestive look. The presence of gall-stones in this case was of interest, as pointing to a local origin of the disease, since primary carcinoma of the gall-bladder is excessively rare.

Dr. Musser replied that Murchison teaches that primary cancer of this organ is always associated—probably causatively—with the presence of gall-stones. *May 26th*, 1881.

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19. *Enlargement of Peyer's patches after pork poisoning.*

By Dr. M. Longstreth.

The history of the specimen was as follows.

The patient was a young child, about three years old. She had always been healthy until the present attack. A few days before death she had partaken of some ham, together with other members of the family, who were also made ill by it. The ham was obtained by the father of the child from a supper given by a society of which he was a member, and of which he was the committee of arrangement for the supper. The ham had been partly eaten at the supper by the guests, and the rest he had carried home to his family. None of the guests, so far as is known, were made ill by eating the pork. The mother of the child said that the ham appeared well cooked and seemed to be good. None of this meat could be obtained for examination. Shortly after eating portions of the ham nearly all the members of the family suffered some ill effects, resembling in kind, though of less degree, than those of the child whose intestine was presented.

The symptoms of this infant were vomiting followed by purging, fever, restlessness, and crying and moaning. The abdomen was swollen and evidently tender. The stools were mostly mucous in character. Death followed on the second day after the beginning of the symptoms.

Another child, younger than this patient, died two days later. It

had eaten less of the ham. Its symptoms were of the same character. No post-mortem examination was made of it.

*Post-mortem Examination.*—No changes were found in any organs except the stomach and bowel. No evidences of peritonitis were found. Both the stomach and intestines were very much distended both with gas and fluid. This fluid was of a whitish color and opaque. In it floated small undigested fragments of coagulated caseine from the milk, but the bulk of the fluid, which was very large in quantity, was chiefly composed of mucus.

The gastric mucous membrane was covered with thick adherent mucus, and its tissue was markedly swollen. The intestine, however, showed these conditions in a more marked degree, especially the lower portion of the ileum, where the villi presented a velvety appearance, owing to their swelling and enlargement. Nearly all of Peyer's patches were swollen and elevated about the surface, in the same manner as seen in the early stage of typhoid fever. At no portion, either of the intestinal or of the gastric mucous membrane, were there any appearances of corrosion or of ulceration. The mesenteric glands were enlarged, especially those in the segment of the mesentery attached to the lower portion of the ileum. *February 24th*, 1881.

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20. *Enlargement of Peyer's patches in measles.*

By Dr. M. Longstreth.

The specimen was taken from a child, aged seven years, dying of catarrhal pneumonia following measles. The patient, Christiana Emil, was admitted to the Pennsylvania Hospital April 18th, 1881. Twelve days previous to her admission, while on the voyage from Liverpool, she, with her two brothers, was attacked with measles. On admission, the fever had subsided, the eruption disappeared, and desquamation was nearly completed. The child was very weak, and had a cough, with abundant mucous râles on both sides of the chest. A few days later, there was a sudden rise of temperature, which subsided nearly to the normal on the day following. This was succeeded by increased difficulty of respiration. Death took place on May 2d. During the time she was in the hospital there had been no symptoms in connection with the digestive mucous membrane to excite any comment.

The autopsy showed catarrhal pneumonia of both lungs and a right-sided pleural effusion. All of Peyer's patches and numerous solitary follicles were enlarged and projected above the surface of the mucous membrane. The other portions of the intestinal mucous membrane showed slight catarrhal changes, not sufficient, however, to give rise to a swollen appearance. A few of the mesenteric glands were slightly swollen.

These specimens, taken in connection with the intestine of the infant supposed to have died of typhoid fever, whose case was presented to the Society in February, raised the interesting and important question of whether other diseased conditions than typhoid fever may not have changes in Peyer's patches similar to those which are considered pathognomonic of enteric fever. This question Dr. Longstreth considered so important that any light thrown on the subject from pathological research could not fail to be of great interest and value, and to this end he requested his friend and assistant, Dr. Henry Dickson Bruns, of New Orleans, to aid him in the investigation. Dr. Bruns's careful researches in connection with leprosy, and his faithful work in pathological as well as other studies, give to his investigations of this question, which are here presented, a high value. Dr. Longstreth took pleasure in giving the paper prepared by Dr. Bruns, in full.[1]

*February 24th*, 1881.

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21. *Specimens from a case of obstinate constipation of eighty-seven days, caused by a cancerous stricture of the descending colon.*

Exhibited by Dr. E. P. Bernardy.

The patient, male, aged 25, previous to his last illness had always enjoyed good health. About a year ago, after eating a hearty meal, he lay on the floor, and while in this position a child four years old fell from the bed, striking him on the abdomen. This was followed in an hour by a free intestinal hemorrhage, which proved obstinate and only yielded after a week's treatment. Dating from this time, he suffered more or less from colicky pains, accompanied with constipation, which were relieved by purgative medicine. On July 19, 1880, after suffer-

[1] For Dr. Bruns's paper, see page 294.

ing for a week, and after taking several purgatives to relieve him, finding himself worse, he called in his family physician, who placed him on small doses of mass. hydrarg., ext. belladonnæ, and ext. nucis vomic. This treatment was followed by relief for a few days, when his sufferings returned with increased intensity, and constant vomiting of a bilious matter supervened. The pills were changed to powders of calomel and morphia. On July 31st his bowels were spontaneously opened, making the nineteenth day from his last passage. The abdomen now commenced to be tympanitic. The rectum was then made to take the place of the stomach, beef-tea and brandy injections being ordered.

I saw the patient for the first time on August 1, 1880. He was somewhat emaciated; pulse 120; tongue dry, but not coated, looking somewhat red and irritated; with a temperature of 101°. The abdomen was enormously distended and tympanitic; in the *right inguinal* region was an indurated mass, dull on percussion; there was no pain on the left side. On account of the indurated mass on the right side, I was somewhat dubious as to my diagnosis, but was of the opinion that I had to deal with a stricture. I simply ordered chloral hydrate with morphiæ sulph., and externally flaxseed poultices. By some misunderstanding, I did not see the case again until September 10, 1880, when I was requested to meet Dr. Hooper in consultation. The condition of the patient was in every way worse; all the above conditions were increased, the spasm of the bowels could be distinctly seen through the attenuated abdominal walls, and everything was rejected from the stomach. The indurated mass in the right side had increased in size, having the appearance as if it were about to ulcerate. All medicines by the mouth were stopped, and hypodermic injections of morphia ordered.

The condition of the patient was the same, with slight variations, up to October 1, 1880, when word was sent that the bowels had been opened; but on examination it proved to be the accumulated residue of the beef-tea injections. On the 5th of October the patient suffered greatly from tenesmus. On examination a large hemorrhoid was found protruding. I then passed my hand up the rectum, but detected nothing. Two days after, I repeated the examination, and detected through the walls of the rectum, on the right side, a large tumor, which filled the whole top of the pelvis; it could easily be indented, but it could not be pushed aside. This on post-mortem proved to be the greatly distended lower portion of the ascending colon. Examina-

tion of the left side revealed, as I thought, a stricture just at the superior strait. On October 11, 1880, Dr. Curtin was called in consultation. The patient was then suffering the most agonizing pain. Dr. Curtin confirmed my diagnosis. A few days after, to relieve the patient from the abdominal distension, I passed an aspirator needle through the abdominal walls, which gave vent to a large quantity of air.

On Saturday, October 16, 1880, Dr. Parrish saw the case with me. The patient was very low, and Dr. Parrish suggested the formation of an artificial anus. This I had proposed on my second visit, but was opposed by the family; but I doubt if the operation would have been of any benefit.

The patient gradually continued growing worse. Tuesday afternoon, October 26, 1880, he had a slight passage of a clay color and very offensive. This was the eighty-seventh day from his last passage. On the morning of October 27, 1880, he passed nearly half a chamberfull of the same colored feces. His pulse was then 140, he was semiconscious, and was evidently dying. Death took place on the morning of the 28th of October, 1880, after the most excruciating suffering.

*Post-mortem.*—Present, Drs. Hooper, Parrish, Allis, and Bernardy. Dr. Allis conducted the autopsy, which was made about six hours after death. Body greatly wasted, with every appearance of death from starvation. Abdomen distended and tympanitic. An incision was made from the ensiform cartilage to the pubes, and a second along the lower border of the ribs; this brought all the intestines directly into view, greatly distended, and of a dull silver color, with the arteries greatly enlarged. Both the small and large intestines were dilated, the latter the most. The usual displacement in such cases was present, viz., a letter U displacement of the transverse colon. A remarkable feature in the case was the pushing up of the liver, so that its lower surface was on a level with the nipple and about five inches from the lower border of the ribs.

The position of the constriction was in the left iliac fossa, above the sigmoid flexure, and seventeen inches from the anal orifice.

The kidneys and liver were examined, but no disease was apparent. The stomach was somewhat contracted, and pushed up under the sternum.

The examination was confined to the abdomen.

*April 14th*, 1881.

22. *Carcinoma of the lower portion of the œsophagus and the stomach.*

Presented by Dr. E. T. Bruen.

The specimen affords an example of cancer of the œsophagus at its termination in the stomach, and the growth has involved the tissue of the cardiac end of the stomach, along its greater curvature more especially. The mucous membrane of the stomach is still intact, although it bulges inward from the presence of the growth, the masses of which can be felt as large as though two or three walnuts were encased beneath its folds. The spleen is found tightly bound to the stomach, the left lobe of the liver as well, and the lung was also fixed. This is shown by the fragments here exhibited. Almost the natural lumen of the œsophagus is preserved. I could introduce my finger with ease before I opened the tube. This I had inferred before death, because I could introduce a fair-sized bougie into the stomach, and this was several times accomplished during life.

All the other tissues of the body were found at the autopsy to be normal. The interesting clinical facts connected with this case can be briefly stated as follows:—

1st. There was no evidence of previous cancer in the family history of the patient.

2d. Although dyspepsia and failing strength had been noticed for six months before the case came under my care, yet all this time the patient was not prevented from attending to her occupation as washerwoman, and she did not apply for treatment till about six weeks before her death, when obstinate, uncontrollable vomiting rendered her unable to work, and she entered the hospital. The material vomited never contained blood, and we now recognize that there is no ulceration of the mucous membrane of the stomach.

3d. The only dulness was furnished in the normal splenic area, and the tumor was situated so high in the left hypochondrium that palpation was equally futile: so no assistance was furnished by physical examination.

4th. Much flesh was lost during the six weeks of vomiting; but little had been lost before, although the patient admitted that she was conscious of failing strength for the six months previous to admission to the house.

5th. The age of the patient was 58 years.

6th. There was some albumen in the urine, and its specific gravity was low (1009), and the question, "Can the vomiting be symptomatic of deficient renal activity due to cirrhotic changes?" was entertained. The hypothesis was, however, not supported by any hypertrophy of the heart or evidence of increased vascular tension.

7th. In forming a diagnosis in the absence of positive symptoms, I could only infer that cancer existed.

8th. The adhesions presented by this specimen are of interest in studying cases of cancer of the stomach. I have now a case in my wards in which adhesions have apparently been formed between the pyloric end of the stomach and the under surface of the liver. Thus a possibility of cancer of the left lobe of the liver is simulated. In such a case the pylorus, weighted by the growth, is sometimes quite movable, and can thus be distinguished from a cancer of the liver, and yet sometimes the adhesions may prevent much movement: so in such a case it can cause pressure on the aorta, and a murmur which is influenced by position.

I should like to pursue this subject, but believe that this is not in conformity with the scope of this Society.

Dr. Tyson said that the case was interesting to him chiefly from the fact that the carcinomatous growth projected outwards, instead of encroaching upon the lumen of the stomach, as is usually the case.

Dr. Henry remarked that the case just reported opened up a very nice question in diagnosis. It will be recalled that the urine was of low specific gravity—1009—and contained albumen, but its diurnal amount was not mentioned. This abnormal condition of the urine coexisting with obstinate vomiting, it was natural, in the absence of *signs* of gastric disease, to attribute the condition of the patient to a diseased state of the kidneys; but Dr. Bruen was led, chiefly, he believed, from the extremely obstinate character of the vomiting, to suspect, and, as Dr. Henry understood him, to diagnosticate, a carcinoma of the cardiac end of the stomach. Bright's disease had been excluded on account of the absence of cardiac hypertrophy and increased vascular tension. Cardiac hypertrophy, although the rule in cases of contracted kidney, is by no means invariably met with. For its development a good state of nutrition is essential. This fact was illustrated by a specimen of extreme cirrhotic atrophy of the kidney, associated with phthisis, recently presented by Dr. Henry to the Society. There was no cardiac hypertrophy, the low state of the patient's nutrition forbidding this compensatory action on the part of nature.

On the contrary, the heart was somewhat below the average normal size, of flabby consistence, and somewhat fatty. Dr. Henry did not recollect whether Dr. Bruen had made any particular mention of the post-mortem appearances of the heart and kidneys in the case just reported.

In reply to Dr. Henry, Dr. Bruen said that the statement that the other tissues and organs were normal had been overlooked: this, of course, included the condition of the cardiac muscle. Dr. Bruen did not think Dr. Henry's case of phthisis with granular kidney, yet without cardiac hypertrophy, a parallel case with that under discussion. In his case the general nutrition had been good up to within a few weeks of the death of the patient, while Dr. Henry's was a case of chronic wasting disease.

Hypertrophy of the heart with accentuation of the sounds, especially the second, is recognized by Dickinson and others as most frequently associated with renal fibrosis. In his clinical experience these facts were cardinal points of diagnosis. If, as occasionally happens, the patient suffers from wasting disease, these conditions may not be present, but even the dilatation of the heart is probable. He considered his point well taken,—viz., that the absence of accentuated sounds and cardiac hypertrophy excluded any renal alteration, for the wasting had been rapid, not chronic. *November* 11*th*, 1880.

## III. THE VASCULAR SYSTEM.

### 1. *Two specimens of aneurism of the thoracic aorta.*

Presented by Dr. E. T. Bruen.

The clinical history of the first case which I shall present will be found in the *Medical Times* for October 11th and 25th, but the specimens, I hope, will interest the Society, and I, therefore, present them with a brief description. Specimens taken from the body of George W., æt. 38 years.

Examining the aorta, a globular tumor as large as a goose-egg will be found arising from the superior portion of the right side of the ascending aortic arch. This tumor grew outwardly, causing a swelling, apparent during the patient's life, between the second and fourth ribs on the right side, at their junction with the sternum, and extending laterally three and one-quarter inches to the right side from the median line.

From the posterior portion of the ascending arch another tumor, similar in size and shape, is found. This tumor extended backward until it touched the bodies of the vertebræ, pressing inward the trachea, decidedly diminishing its calibre, just above the point of the bronchial bifurcation. The tumor is also adherent to the right bronchial tube which leads to the inferior lobe, almost occluding its lumen; the union between the tissues of the two structures is so intimate that I cannot separate them.

The inferior portion of the lower lobe of the right lung is collapsed and splenified; the remaining portion of the pulmonary tissue is emphysematous, completely covering the splenified area.

Passing over the inferior surface of the lower tumor the pneumogastric nerve can be seen; during life this nerve must have been compressed between the tumor and the bodies of the vertebræ.

The tumors are very hard and solid; when I introduce my finger into them they are found almost completely filled with organized fibrin, so that but little blood could have entered them. During life no thrill

could be felt over the tumor, and only a feeble systolic murmur could be heard, which was intensified by pressure.

The cause for the absence of these physical signs is now clear, since no blood could have entered the tumors. The increase of the murmur by pressure is explained, I think, by the fact that such pressure must have diminished the calibre of the aorta, just as a murmur can be developed in any superficial artery.

It seems to me that if this explanation of the clinical observation had been appreciated during the life of the patient, it would have led to the diagnosis of the contents of the tumor which was superficial.

The radial pulse during life was noted to follow the femoral pulse; this must have been due to some interference with the passage of the blood into the innominate or subclavian arteries, but the tumors do not appear to obstruct their orifices in any way.

Death resulted from asphyxia due to pressure on the pneumogastric nerve and bronchial tubes.

There was no admission of syphilis on the part of my patient, but he confessed that for many years he had used alcoholic stimulants to excess.

In the second case the patient was admitted to the Philadelphia Hospital on the evening of Saturday, October 18, 1879.

She was almost moribund from asphyxia, evidently in part due to some laryngeal obstruction, in part to intrathoracic pressure on some portions of the bronchial tubes. She died twenty-four hours afterwards. She had been an inmate of the house several years before, suffering at that time from rheumatism; but there is no record as to whether or not this was syphilitic. She was 43 years old.

On examining the aorta, a number of small saccular pouches are found,—one at the junction of the descending arch with the portion of the aorta called thoracic. This sac was adherent to the vertebral column, and had eroded very deeply the bodies of two vertebræ. A second sac projected backward from the descending arch, compressing the œsophagus. Here the coats of the aorta have disappeared, and the blood is separated from the œsophageal tube only by its own walls.

Another sac bulges the transverse arch anteriorly. The entire aorta is covered with atheromatous plates. The valves of the aorta are sufficient; the heart is somewhat hypertrophied. The lower lobe of the left lung is splenified; the lower lobe of the right lung is solidified partly by splenification, partly by pneumonia.

There is no *direct pressure* on either *bronchial tubes* or trachea—as

the specimen lies before me. During life, however, it is most probable that these sacs, being distended by blood, did occasion pressure on both the trachea and the bronchial tubes.

Moreover, two of these sacs arise near the position at which the recurrent laryngeal nerve of the left side passes around the aorta; doubtless the nerve was irritated, causing laryngeal spasm.

These tumors are the smallest I have ever seen associated with such severe symptoms during life, and with the pulmonary conditions I have described.

In these two cases we have the illustration that emphysema, splenification, and pneumonia can be caused by intrathoracic pressure from aneurism of the aorta. *October* 23*d*, 1879.

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## 2. *A case of paracentesis pericardii.*

Presented by Dr. Comegys Paul.

In his "Practice" Professor Flint has remarked that "the clinical data for determining the value of the operation of paracentesis of the pericardium are wanting." The few recorded cases in this country of an operation suggested at a time when medicine was not so exact a science as it is now, fully warrant such an assertion. An examination of authorities reveals the rather general opinion of doubtful expediency, most of them, however, admitting that it may *sometimes* be resorted to with benefit. It would seem that a want of success has exerted an influence very similar to that which controlled our judgment in regard to tracheotomy not many years ago; and it is not improbable that it will soon be understood that this operation, like tracheotomy, has been unsuccessful because of its delay until the vital power became exhausted, and not because of any bad effects from the procedure itself. As a purely mechanical remedy, it certainly has as much to recommend it as venesection practised for the relief of an overloaded heart; and when our predecessors let blood in cases of sthenic pneumonia, to restore equilibrium to an obstructed circulation against which an embarrassed heart was laboring without avail, it would have been as plausible to attribute the fatal result to venesection as to claim that death is due to paracentesis rather than to the hydrostatic pressure of a distended pericardium.

The interest attached to this subject has been renewed by the recent

discussion of the Philadelphia County Medical Society and the publication of an interesting and successful case by Dr. Pepper.

The relation of the following case is made because of several unusual circumstances attending it. The pericarditis was idiopathic, and failed to furnish as its cause any of the diseases usually considered as such. Suspecting a latent albuminuria, the kidneys were carefully examined; but they showed no trace of inflammation, nor did the lungs or pleura, except a small patch of hepatized lung, which can be otherwise explained. Neither was it a sequel to any of the continued and eruptive fevers. It is hardly necessary to say that there was neither rheumatism nor any traumatic reason.

Augustus H., a convict, 22 years old, of good physique and well nourished, had been in close confinement for nearly a year. With the exception of what is known as "prison pallor," there was no indication that incarceration had interfered with his general health. On the 18th of September he complained of an indefinite sense of distress, with slight fever and a suppressed cough. Respiration was somewhat increased and the pulse accelerated. Pain in the præcordial region was influenced by inspiration, which became shortened for this reason, as often happens in attacks of pleuritis. Physical examination located the disease in the præcordia by an unmistakable pericardial or cardiac friction-murmur.

The next day abnormal dulness appeared in this region over an increased area, with unusual prominence of the intercostal spaces. The apex-beat had been forced beyond the nipple, and the heart-sounds were more or less muffled and diminished in force. The patient now wore an anxious expression of countenance, and was more comfortable in a semi-recumbent position. The pulse fluctuated between one hundred and thirty and one hundred and forty beats, and, with the respiration, was easily excited by the slightest muscular exertion, causing at times a marked dyspnœa.

For the next three days these symptoms became more aggravated, the dyspnœa becoming continuous. The extremities were now cold as far up as the knees and elbows, and the stomach refused nourishment. Inability to sleep except in short naps, a constant tossing about of the body, a painful anxiety, and fear of death, were other indications of its near approach.

At this juncture I debated with myself whether the time had gone by when paracentesis pericardii would be of any avail in warding off the fatal result. As a last resort, I believed it to be practicable in

this case, and, in the presence of Dr. E. C. Hine, and with his concurrence, I decided to adopt it. Not having an aspirator or a small trocar, necessity obliged me to make use of a long and large hypodermic needle. The puncture was made in the fourth intercostal space, half-way between the nipple and the middle of the sternum, and directly over the right ventricle. Half a syringeful of serum slightly tinged with blood was withdrawn, when the needle became clogged. It was not deemed expedient to repeat the operation with such an imperfect instrument, and no benefit was expected to follow it. Within two hours after, and contrary to hope, the præcordial oppression diminished, the circulation improved, the blue color of the lips disappeared, and his hands and feet became warm. This unlooked-for reaction continued during the next day and the day following. On the third morning his pulse was 90 and of fair strength. Towards evening he became worse, a relapse followed, and he died at three o'clock on the morning of the fourth day after the operation and the ninth day of the attack.

*Autopsy.*—Upon opening the chest, a quantity of serum was found in the mediastinal space, and a little in either pleural cavity, perhaps a pint in all. Neither the parietal nor pulmonary pleura exhibited any trace of inflammation. The lungs were of normal appearance, except a very small and circumscribed portion of the lower lobe of the right lung, about the size of an infant's hand, which overlapped that side of the heart. This inconsiderable and almost insignificant part was hepatized by vicinal congestion or injured by the needle. The puncture of the needle had not left a discernible wound in the sac, but on the surface of the wall of the right cardiac ventricle were three minute and superficial scratches. These accounted for the bloody tinge of the serum within the pericardium. The pericardial space was much exaggerated, the pericardial sac containing about eight ounces of fluid and surrounding a heart hypertrophied to nearly twice its natural size.

The abdominal organs were in a natural condition.

This case may not be of less value because of its fatal termination. The post-mortem appearances afforded me satisfaction not only because they confirmed the diagnosis, but because the relief that followed the operation was in a measure explained by them; and on this account it would seem that the clinical facts have assumed more importance than if the result had been otherwise.

From the meagre quantity of fluid directly withdrawn by the in-

strument, we cannot in any way suppose the patient to have been benefited. The fourth intercostal space was chosen for the puncture because it appeared to be and was more directly over the right ventricle in this case, although Roger and Dieulafoy recommend the fifth. According to Dr. J. B. Roberts, the pleura must be wounded, "because it is reflected over the pericardium from the costal cartilages;" and I think the extreme border of the lung is unlikely to escape. The quantity of serum in the mediastinal space and in both pleuræ by endosmosis is evidently the explanation of the gradual relief that followed the above operation. The over-distended pericardium allowed the confined liquid to escape slowly through the puncture, thus gradually removing, in two hours, the powerful compression under which the heart was laboring. This relief was continued for nearly four days, when, the aperture having become obliterated, the pericardium slowly, but partially, refilled itself. The prostration of the patient was unequal to the resistance of a renewed attack, which obliged him to succumb finally to asthenia. *Oct. 23d*, 1879.

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### 3. *Aneurism of the innominate artery.*

Presented by Dr. J. H. Musser.

As Medical Registrar at the Hospital of the University, I had the opportunity of observing the patient from whom the specimens were taken, and through the courtesy of Professor Pepper I am allowed to present them to you.

Admitted to the hospital August 1, 1880, the patient was 34 years of age, a native of Wales, and a resident of Dauphin County, Pennsylvania. For twelve years past his occupation has been that of a miner, working hard and exposed to all hardships. A constantly moderate drinker, at times he would spree. Always used tobacco moderately. Eleven years ago had a sore on the penis, said to be a chancre. A year afterwards he had subacute rheumatism, lasting a year. No other symptoms of syphilis. Save an attack of pneumonia, he has been perfectly healthy. Family history, as far as can be ascertained, good.

While working in the mines when they were on fire, in January, 1879, he caught cold, and thenceforth suffered from shortness of breath, stiffness in the neck, and a "twitching" sensation in the larynx; the

latter symptom occurring at intervals, the others being constant, though variable in degree. In January, 1880, he applied to a physician for treatment. At that time he was said to have bronchitis and chronic laryngitis. Early in July he had an attack of what was said to be œdema of the larynx. A few days afterwards a "lump" broke in the throat, with the discharge of purulent matter. Since then he has expectorated blood. After the breaking of the "lump" the dyspnœa was relieved. During the year he has lost flesh and strength. His appetite has been poor, and he has had night-sweats. At present he is a moderately stout man, with a large frame, weighing one hundred and sixty-two pounds, face slightly flushed, not anæmic, skin warm and moist, fingers not clubbed, dark eyes, long eyelashes, hair in abundance, dark, teeth good, natural countenance, tongue coated and flabby, pharynx slightly congested, no blood on it or point of hemorrhage, tonsils and half-arches normal, no dyspeptic symptoms, bowels regular.

Laryngoscopic examination by Dr. McCracken, in charge of throat department, revealed only slight infiltration of the tissues about the glottis, absence of paralysis of either vocal cord, in the trachea at the right a mass of blood. Did not notice any occlusion of the tube. At the larynx there is a hoarse, coarse inspiratory sound; the sound is transmitted all over the thorax, so that it is almost impossible to hear the respiratory murmur. The percussion-note at the left apex was a little higher-pitched than normal. Expansion of the apex and of the whole lung was poor. Constant cough; continuous expectoration of bright arterial blood. Pulse 70, heart normal, special senses and mental faculties unimpaired.

Further note.—For three days the hemorrhage continued; on the third day death resulted from a sudden enormous hemorrhage.

*Post-mortem.*—All the tissues and organs, except the lungs, exsanguinous. The organs were healthy. The lungs, air-cells, and bronchii filled with arterial blood. On opening the thorax, the heart and lungs were in their normal positions. The inner sides of sternum and ribs were not pressed upon. The innominate veins were normal in position and size. The trachea was not noticeably deflected. The ascending and transverse portions of the aorta were dilated, the upper part being more expanded. The innominate artery was dilated, the dilated mouth being continuous with the dilated aorta. The expansion of the innominate was not uniform; anteriorly there were one or two sacs, posteriorly a large sac pressed against, became united to

and ulcerated into the trachea. The right carotid arose from the common expansion of the aorta and innominate, the subclavian one and a half inches from the carotid, above and at the right of the mass. The dilated portions of the aorta were atheromatous. Within the aneurism, completely filling it, was a large, moderately firm, pale-red clot; left ventricle of heart slightly hypertrophied; valves normal. I should have mentioned that the pneumogastric nerve was seemingly much pressed upon, and the cords of the plexus were also quite flattened where they passed over the mass. The recurrent laryngeal was not pressed on.

The diagnosis was not made before death. It was thought to be incipient phthisis with tumor. I thought there must be something causing pressure. I had no opportunity for a second examination. Had more careful percussion been performed, the dilated aorta and the aneurism could probably have been defined. Comparison of the radial pulses might have given a clue. There was no projection, no pulsation, neither thrill nor murmur, nor were there any external evidences of pressure within.

The continuous dyspnœa, persistent stiffness of the neck at the root, with soreness, and the "twitching" sensation, are worthy of note. The July attack was due to œdema probably, and the "breaking of the lump," to the completion of the ulceration, hemorrhage being prevented by the protruding clot.

I am positive that the circulation of the right arm was not interfered with nor the pulse much altered, as it was at that arm I noted the pulse.

Dr. Bruen had for some time past in the study of aneurism taken as fundamental evidence for its diagnosis two series of facts, viz.: 1, the sequence of disease of the arterial coats upon some prior cause capable of producing inflammation of the arterial coats; 2d, symptoms of pressure. The pressure-symptoms seem to have been overlooked in this case. It was, indeed, true that other diseases besides aneurism might give similar pressure-symptoms; but, as a rule, they could be differentiated easily. He knew of a case in which an erroneous diagnosis of dilated aorta had been made, when fifteen months afterwards mediastinal cancer was undoubted; cases of this sort are exceptional.

Auscultation and palpation were not so important, in his estimation, as the above points, for a deposition of laminated clot might prevent murmur, thrill, or pulsation. In proof of this, he would recall to the remembrance of the Society a case reported by him to this Society about

one year ago,[1] in which case multiple aneurisms had been found after death, completely filled with laminated clot, so that no blood could enter them. During life, murmurs could be elicited only by pressing the most superficial tumors upon the aorta, thus obstructing its lumen. In these cases, the pressure-symptoms, aided by percussion, were most interesting.

Two cases of abdominal tumor which he had lately seen presented the same symptoms as regards murmur and palpation signs of tumor. In one there was a specific history, and a peculiar pain relieved by a position of the body prone upon the face. In the other there is no history of syphilis, alcoholism, or renal disease, nor is there the significant pain relieved by change of posture.

In one of these cases the diagnosis was verified by autopsy, revealing rupture of an aneurismal sac; the other, Dr. Bruen believed, will be demonstrated at an autopsy to be carcinoma, probably of the stomach, and the diagnosis was made by attention to his cardinal points. In abdominal carcinoma adhesions might form, which might so fix the tumor as to cause a murmur not affected by change of position.

Dr. Musser, in reply to Dr. Bruen, called attention to the fact that he had stated in his notes that perhaps more careful percussion would have detected the tumor and the dilated vessel. Neither, however, encroached upon the lung, as that organ was most carefully examined. The anterior view of the tumor was small, as dilatation of the aorta was not very great, so that the area of dulness would certainly have been small. Pressure-symptoms were noted, but the sudden death of the patient prevented the cause from being investigated fully. The stridulous respiration, its seat loudest in the larynx, the paroxysmal attacks of dyspnœa, finally growing constant, were due, probably, to the stricture of the trachea.[2] Œdema of the glottis might have been produced by obstruction to the venous return from pressure. The flattened condition of the right pneumogastric nerve should have caused some more constant symptoms. The paroxysms of dyspnœa might have been thus produced.

Dr. Bruen asked if the dyspnœa was spasmodic or constant.

Dr. Musser replied that at first it was spasmodic, but latterly it was very slight, but constant.

[1] Meeting of October 23, 1879; also Medical Times for October 11 and 25, 1879.

[2] Ziemssen's Cyc., art. Occlusion of Trachea.

Dr. Bruen referred to a case of spasmodic dyspnœa resembling asthma, which was due to aneurism. He had already this evening referred to this case. The aneurismal growth had compressed the pneumogastrics and trachea, and produced the same whistling respiration noted in Dr. Musser's case, and horrible asthmatic seizures, although there was always some dyspnœa. In Dr. Musser's case these symptoms would probably have become more marked as the tumor enlarged.

Dr. B. also wished the Society to note that he had also read before them a paper (afterwards published in the *Medical Times* of May last) on mitral obstruction, in which he had claimed that murmurs indicative of mitral obstruction were heard with maximum intensity over the auricle, following the ordinary law, that murmurs are heard with maximum intensity at that point where the cavity in which they are produced approaches closest the surface of the chest. This, with accentuation of the second sound at the pulmonary artery and enlargement of the auricle, often helps to disentangle a confused murmur.

Dr. Musser thought these symptoms were due to the pressure on the trachea, especially as the same dyspnœa, early in July, was relieved by the "breaking of the lump," which had doubtless lessened the tension of the aneurismal sac. He would also call attention to the constant soreness and stiffness at the root of the neck.

Dr. Tyson asked if any disease of the lungs had been detected post mortem.

Dr. Musser replied that none had been observed.

*Nov.* 11*th* 1880.

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4. *Mitral, aortic, and tricuspid valvular disease; red atrophy of the liver; cirrhosis of the kidneys and pancreas.*

Presented by Dr. J. H. Musser.

The specimens I present are from a patient who died in the University Hospital. The history is as follows:—

Maggie M., white, æt. 26, single; born in Dublin; but three weeks in this country; domestic. Father and mother living; six brothers and sisters dead, cause not known by patient. No cancer or phthisis in the family to the patient's knowledge. Five years ago had acute rheumatism; no other illness.

September 22d of this year she was suddenly seized with vomiting

and slight fever. In two days the vomiting was replaced by pain over the xiphoid cartilage. On the 27th she became jaundiced. The above is the patient's story of her condition previous to admission. I may say she drank daily of ale and porter, occasionally to excess.

October 2d, her condition was as follows: emaciated and anæmic; skin and conjunctivæ jaundiced. Face dusky, lips blue, countenance anxious; hands and feet blue and cold, fingers clubbed; temperature 95°; mind dull. Tongue dry, heavily coated with a dark fur; thirst; no vomiting; loss of appetite; pain and tenderness upon pressure on epigastrium. Liver dulness in mammary line begins at fifth rib and extends to the margin of the ribs. In median line, dulness extends to two inches below xiphoid cartilage. Slight diarrhœa, stools light yellow. Breathing hurried; lungs healthy; cardiac impulse in second, third, and fourth interspaces, one, one and a half, and two inches, respectively, inside the nipple-line, and in the fifth interspace one-half inch outside nipple. Palpation shows same as inspection, and in addition an epigastric impulse is noted, and at the apex a slight thrill. Cardiac dulness begins at second interspace above. The right border corresponds with the right edge of the sternum, the left with the left limit of the impulse-area. Heart-sounds extremely tumultuous and difficult to define. The first was somewhat booming, with a murmur over the mitral area. At the aortic cartilage a feeble systolic sound, possibly transmitted; pulse very small, weak, irregular, about 160. Urine: reaction, acid; specific gravity, 1021; albumen present; sugar absent; sediment, few granular and numerous hyaline casts, some of them containing red blood-corpuscles; fatty epithelial cells from the bladder and urethra, some of them stained yellow; few pus-cells; irregular granular masses of brown pigment and broken-down red blood-cells.

October 3d and 4th, condition about the same. Later, the night of the 4th, she had an attack of dyspnœa, and of severe pain over the sternum and in the right side. There was dulness at the base of the right lung, and subcrepitant râles. Digitalis and carb. ammonia, with dry cups, gave some relief. At 4 A. M. she almost went into collapse; her extremities were cold and blue, face dusky; pulse imperceptible at wrist; heart very rapid, weak, and irregular; subcrepitant râles and dulness at the base of left lung, also. Internal and external stimulation caused relief, so that in the morning she had only slight pain and oppressed breathing; extremities were warmer, face more natural, although jaundice seemed increased; pulse was 150, stronger

and more regular. Congestion of lungs posteriorly continued; slight diarrhœa, pale stools; no vomiting.

October 5th, 4 A. M., another attack of dyspnœa. At the morning visit the pulse was improved slightly, as was also the dyspnœa; jaundice slightly increased. In the afternoon she became dull, not answering questions readily; heart as usual; no dyspnœa; urine not passed so abundantly as in health.

October 6, weak; breathing hurried; effusion in both pleuræ; heart's action very rapid and feeble, irregular; pulse not felt. Death at 10 A. M.

Dr. Formad made the following report of the post-mortem, made by Dr. Hughes, three and a half hours after death :—

External appearance: whole body jaundiced; large yellow patches, especially about the head; sclerotic coats of eyes of a deep-yellow color; pupils dilated; abdomen slightly distended; rigor mortis well marked.

*Thorax.*—Pericardium contained about six ounces of a straw-colored fluid; about two pints of a similar fluid in each pleural cavity. Firm lateral and diaphragmatic pleural adhesions of left lung. *Lungs* congested; no œdema; crepitant throughout. *Heart* somewhat enlarged, weight fourteen ounces; muscle dark; coronary vessels engorged. Pulmonary artery and valves normal. Tricuspid valves thickened and rigid, orifice contracted. Right auricle and ventricle slightly dilated and filled with dark-red clots and a chicken-fat clot. Aortic orifice was much narrowed by the stiff, retracted, partly calcified leaflets. The valves could not have effected a sufficient closure. Mitral valve-leaflets diseased, permitting regurgitation; the orifice was narrowed. Columnæ carneæ thickened and rigid. On the endocardium of the left ventricle, at the apex, and near the mitral valve, small white patches of lymph-like appearance were seen.

*Abdomen.*—Only a small amount of effusion in cavity. Diaphragm and organs in proper position. *Omentum* normal. *Spleen* diminished in size, very hard. *Pancreas* increased in thickness and weight, cirrhotic. The head was so enlarged that it must have pressed on the common bile-duct. *Gall-bladder* distended and filled with bile; duct pervious. *Liver* diminished to about two-thirds its size, weighing one pound thirteen ounces, left lobe somewhat enlarged, however; firm on pressure, surface unevenly yellow tinged, on section "nutmeg" appearance characteristic. *Stomach* of normal size, in greater curvature mammillated. *Kidneys* normal to naked eye, congested, capsule read-

ily removed. *Suprarenal* capsules thickened, hard, and containing white nodules. *Bladder* and *ureters* normal. On posterior surface of *ureters* a small fibroid tumor. Death was due, no doubt, to cardiac failure. The tricuspid disease was not recognized—perhaps, as Fothergill has suggested, on account of the amount of blood passing through being too small to develop a murmur; especially so because of the altered mitral and aortic orifices. The tumultuous and rapid action of the heart may have prevented its detection. That the murmur was not developed on account of the small supply of blood is most probable, for venous pulse and liver pulsation were absent. Disease of the valve permitting aortic regurgitation was not recognized, from the same cause or causes, or it was entirely overlooked. I think in both cases the murmurs were absent, as most careful auscultation was performed.

On microscopical examination the liver presented the characteristics of red atrophy, the kidneys were slightly cirrhotic, the pancreas markedly cirrhotic. Examination of the white patches in the endocardium proved them to be the result of a chronic inflammation, like that of an atheroma.

Dr. Formad had found, upon examining the pancreas, that it was much enlarged. Microscopical examination, however, showed that this enlargement was not due to any neoplasm, but to cirrhosis and marked increase of the interacinous connective tissue. The white patches upon the heart muscle were due to mere thickening of the investing serous membrane. Portions of these patches were slightly eroded. The liver presented a rare condition, viz., red atrophy. This was most marked around the hepatic venules, where the liver-cells were represented merely by a mass of reddish debris. At the periphery of the lobules the cells were much less affected. These conditions, conjoined with marked cirrhosis, account for the microscopic nutmeg appearance of the organ. The kidneys and the suprarenal capsules were normal.

Dr. Bruen asked Dr. Formad whether there was any catarrh of the gall-ducts.

Dr. Formad replied that he had detected none, the ducts being, however, contracted and obstructed at points.

Dr. Musser asked if the patches upon the heart were due to an acute or chronic change.

Dr. Formad considered the process chronic.

Dr. Henry remarked that it was an interesting point in connection with Dr. Formad's report that the lesions due to obstructed circulation

were so exclusively confined to the liver. He would naturally expect to find in such a case a marked degree of cyanotic induration of the kidneys. *Nov.* 11*th*, 1880.

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### 5. *Aneurism of the cœliac axis.*

Presented by Dr. E. T. Bruen.

These specimens were removed from the body of a colored man, aged about 32, who died in the Philadelphia Hospital a few weeks since. In abstracting my notes on his case I record that a syphilitic history was admitted, but that the individual had been a temperate man in the use of alcoholic drinks.

A pulsation could be felt about two and a half inches below the ensiform cartilage, but a distinct tumor could not be felt. There was no thrill. Careful percussion developed impaired resonance the breadth of the middle finger only. By auscultation, a murmur was detected, very prolonged, low-pitched, and of systolic rhythm. There was no bruit, nor could the murmur be heard in the femorals or at the bifurcation of the iliacs; it could not be heard at the back. It was heard first at ensiform cartilage, grew louder till a point two and a half to three inches below the sternum was reached, and then faded away. A deep-seated burning pain was complained of, and also a scattering pain radiating around to the anterior median line along the ribs: this pain he described as sharp, and at times it was much worse than others. Both sorts of pain were relieved by change of posture. His favorite attitude was lying prone on the face. This helped vastly to mitigate the pain.

A treatment of rest was commenced, and it was designed to place upon him a plaster jacket, commencing the roller high up on the chest and to continue it to the hips. It was hoped that this would, by fixing the aorta, aid the treatment of rest in bed, a restricted and selected diet, and the drugs usually employed in these cases. It was decided to apply this dressing on a Wednesday. The day before, the man rose in the morning, expressed himself as feeling quite well, and was engaged in making his toilet, when he suddenly cried out and dropped dead.

*Autopsy.*—The abdominal cavity was filled with coagulated blood. A saccular aneurism about as large as a good-sized lemon projected

from the left antero-lateral surface of the aorta. The walls of the sac were most attenuated; at the periphery of the tumor the external coat was about as thick as a piece of paper, and very brittle from calcareous deposit. The sac, when laid open, was found to be only partially filled with friable clots, not organized into layers, but irregularly disposed over its walls. The rupture occurred at the point most remote from the aorta, at the periphery of the tumor. The orifice communicating with the aorta was very small, about the size of a large hickory-nut. The other parts of the aorta and the heart were normal. The lungs, liver, and kidneys presented a natural appearance.

I would comment briefly on the infinite importance of quiet and rest in the treatment, and the advantage of fixing the aorta by means of the permanent dressing. Certainly the lateral movements of the body would have been much restricted, and the aorta probably saved many a flexion. Only at the last meeting I spoke of the supreme importance to the diagnostician of the prior history of the conditions favoring arteritis, and the evidence of pressure in the path of the aorta, and the percussion-signs of tumor. In addition to what I then said, let me add a word concerning the murmur as present in this case. The absence of bruit, the prolonged low-pitched murmur with the systole, suggested to me that this was a fusiform aneurism caused by a general dilatation of all the coats of the aorta, especially as the evidence of tumor was so slight so far as percussion was concerned.

The orifice communicating with the aneurism was so small that very little blood probably entered and left the sac; besides, the sac was partially filled with clot, and at the autopsy it lay on the trunk of the vessel, doubtless obstructing its lumen, and I suggest it may have caused the murmur. The ante-mortem inference as to the shape of the aneurism has been, to my mind, sustained by the autopsy.

I show with this case an aneurism exhibited some time ago to this Society, the sacs entirely filled with laminated clot, and in which case there was no murmur, unless the tumor, which projects anteriorly at the second right costal cartilage, was pressed upon so that the calibre of the aorta was lessened. A distant, long, low-pitched systolic murmur, without a taint of bruit, indicates to me that the sac of an aneurism is mostly filled with clots; and, unless I can identify a tumor by palpation or percussion, or by the symptoms of pressure on adjacent organs, I am apt to infer the existence of an aneurism by general dilatation of the coats of the vessel from the above-mentioned murmur. I do not mean to say that these general dilatations do not cause pres-

sure-symptoms, but merely that these are not so pronounced, as a rule. But in this case the murmur deceived me, and a saccular aneurism only half filled with clots exists; but I hope the very small opening leading into the aorta will be deemed a valid reason for the absence of pronounced bruit.

Dr. Nancrede took exception to the term "aortic" aneurism, as he thought that a careful examination of the specimen showed conclusively that it was due to a dilatation of the cœliac axis, and did not involve the aorta at all.

Dr. Bruen thought that the trunk might be partly involved, but was inclined to think that a part of the walls of the aneurism was formed by the aorta.

Dr. Nancrede explained the anatomical reasons upon which he had founded his opinion, and which he thought were conclusive.

The President said that, as there seemed a difference of opinion on the subject, he would refer the specimen for examination to a special committee, consisting of Drs. Bruen, Nancrede, and Formad.

"The committee appointed to examine the tumor agree to pronounce it an aneurism of the cœliac axis rather than aorta proper. This explains the fact that the orifice leading into the aneurism is so small, as it is the dilated cœliac vessel; but remarks in reference to the etiology of the murmur are equally pertinent.

EDWARD T. BRUEN,
C. B. NANCREDE,
H. F. FORMAD."

*December 9th*, 1880.

## IV. THE ORGANS OF RESPIRATION.

### 1. *Intra-laryngeal growth.*

Presented by Dr. C. Seiler.

On June 14th I was asked by Dr. Ludlow to see, with him, a case, and make a careful laryngoscopic examination, with a view to determine, if possible, the cause of a persistent aphonia, from which the patient had been suffering for about eighteen months.

On reaching the house I found the patient to be a gentleman 64 years of age, and elicited from him and from Dr. Ludlow the following history :—

Mr. H., the patient, had always enjoyed good health until about eighteen months before, when, after violent exercise in the open air on a cold winter's day, his throat became sore and his voice hoarse. The usual treatment for acute laryngitis relieved him of his symptoms, with the exception of the hoarseness, which, although not so bad, still continued. He felt quite well until August, 1878, when again his throat became sore and his voice left him. He tried various remedies, and especially counter-irritants about the neck and throat, without relief, and his throat gradually became worse. In the latter part of December of the same year, a swelling of the neck began to show itself, together with shooting pains in the left ear.

Becoming alarmed, Mr. H. consulted Dr. J. S. Cohen, who, on laryngoscopic examination, discovered a small neoplasm overlying the left vocal cord, and advised him to have it removed, but the patient was not then willing to submit to any operation. He afterwards placed himself under the care of Dr. Barton, who made several applications to the larynx by means of a brush, without giving relief. Homœopathic treatment was also resorted to by the patient, at the instance of friends, with no result, and finally he returned to his family physician, Dr. Ludlow. As the swelling on the neck had become very great by this time, counter-irritants and leeches were again resorted to, with transitory relief.

On examination, I found the larynx to be greatly swollen and almost

immovable, but not tender on palpation. The patient complained of a sense of fulness about the throat, shooting pain in the left ear, difficulty of deglutition, together with a dry, short cough, especially during his meals, and dyspnœa. A laryngoscopic examination showed the epiglottis to be swollen and red; the aryepiglottic folds were tumefied so that the outlines of the arytenoid cartilages were obliterated, especially so on the left side. A lobated, raspberry-like growth, of the size of a small pea, was lying on the left vocal cord, and apparently springing from the left ventricle, the ventricular band on that side being tumefied. In the anterior angle of the thyroid cartilage there appeared a small, round prominence, of a white color and smooth, glistening surface, which at the time I supposed to be a small fibrous growth of but little significance. The right side was affected by either the œdematous swelling or the inflammation. A view of the subglottic cavity could not then be obtained.

The patient was then willing to have the tumor removed from the larynx, and, the case being handed over to me for laryngeal treatment, I introduced the guillotine and succeeded in removing a small portion of the tumor. This, however, made the throat very sore, so that I desisted from operating for several days, and ordered leeches applied to the neck. One of the leech-bites discharged pus, and proved, on probing, to be the opening to a sinus some one and a half inches in length.

Several pieces of tumor were subsequently removed, and I noticed that the white swelling at the anterior angle of the thyroid increased very rapidly and encroached upon the opening of the glottis to such an extent that it was deemed necessary to perform tracheotomy, which was successfully done on July 7th, Drs. Cohen, Ludlow, and Wirgman assisting me. The patient recovered rapidly from this operation, and improved in general health so that he was able to be out. The abscess in the neck over the thyroid continued to discharge and the laryngeal tumor to grow more rapidly than the patient would let me remove it, until the pain about the neck became very great and the shooting pain in the ear intolerable; there was also a very harassing, dry cough, which prevented sleep.

Finally, on August 7th, the patient consented to have a free incision made into the abscess, as had been urged by me for some time, and as was advised by Dr. Levis, who had been called in to see the case. The incision which I made discharged but little pus, and, on passing in the finger, it was found that the larynx was open, and a

large piece of papillary growth was removed through the opening. This piece was examined microscopically, and found to be a papilloma, the connective tissue of which was infiltrated, resembling very much sarcomatous tissue, so that the growth was considered to be a sarcoma with papillary outgrowths by Drs. Shakespeare and S. W. Gross, who both examined a section of it.

A slight chill followed the operative interference, which, however, was not repeated, and the patient felt greatly relieved of the pain in the neck and ear, but the cough remained unaltered. The external wound was kept open by a large drainage-tube, and daily small pieces of the growth were removed through the opening. A laryngoscopic examination which was made several days after the external operation showed that the larynx was still filled with the whitish mass, and I then conceived the idea that the piece of growth removed had been external to the larynx, and that the swelling of the neck was in a great measure due to parts of the growth having penetrated the tissues around the larynx. Dr. Ludlow, as well as I, then urged a further splitting of the larynx, in order to remove, if possible, the whole growth, but the patient's family would not consent to any further operation.

The tumor in the larynx continued to grow, and by the middle of October had so encroached upon the epiglottis that deglutition became almost impossible.

The patient then consented to another operation, and on October 24 I performed laryngotomy, Drs. Cohen, Ludlow, Packard, and Wirgman kindly assisting me. The masses of tumor before you were taken, both from within and without the larynx, by means of a spoon and by scraping with the finger-nail, until apparently all traces of growth were removed, and the larynx, as well as the trachea, was found to be clear.

The patient made a rapid recovery from the operation, and in two days was able to swallow solid food; he was able to be out of doors two weeks afterwards. A laryngoscopic examination showed the larynx to be perfectly clear of any growth, but there still existed a stenosis of the trachea just above the tracheotomy-tube, so that the operation had not been successful as regards re-establishing the natural respiratory tract. This stenosis I endeavored to remove by dilatation, passing the dilator both through the mouth and larynx, as well as through the external opening, which I kept open for that purpose. The patient's general health improved very much, and, being desirous

of being able to pass some air through his larynx, he asked that another operation be made in order to remove the obstruction.

This I did on December 6th, Drs. Cohen and Ludlow assisting me, and found that there was a mass of tumor inside as well as outside of the trachea, close down to the opening of the tracheotomy wound, as well as a good deal of cicatricial tissue within the trachea. This latter was broken down, as well as could be done, and the former removed by means of the finger-nail and spoon, until free communication was established between the trachea and the outer opening above.

During this operation, as well as during the former, a novel tracheotomy-tube was employed, at the suggestion of Dr. J. Solis Cohen, in order to prevent any blood from running into the trachea, and also to facilitate the administration of ether during the operation.

The patient made a very rapid recovery from the operation, the febrile symptoms being hardly perceptible, and he was able to be up and about on the next day, but not able to breathe through his mouth, as the wound was still kept closed with cotton with a view to prevent cough from the dropping down into the trachea of pus and blood.

All through the course of the disease the pulse never rose above ninety, except after the second operation, when the patient had the chill, and the temperature also remained normal throughout. There were no glandular enlargements; in fact, no systemic symptoms of malignant growth.

The question of greatest interest in this case is, Where did the tumor originate, in the larynx or outside of it? The appearance of the white swelling at the anterior angle of the thyroid, which was seen in the laryngoscope growing backward and gradually filling up the larynx after the external swelling of the neck had existed for some time, would indicate that the tumor originated outside of the larynx, perhaps in the atrophied thyroid gland, and had pushed its way into the larynx by splitting the thyroid cartilage.

A microscopical examination of sections of the tumor shows it to be composed of pegs of large epithelial cells, many of which are multinucleated, and in the centre of them ducts of glands are seen filled with granular débris. The tissue between the epithelial pegs is composed of young spindle- and round-celled tissue, separating the epithelial elements so that they form finger-like projections.

Dr. Gross said, in reply to Dr. Seiler's reference to his microscopical examination of the tumor, that the section given to him was one obtained from the first scraping of the larynx, and that it presented

the appearance of the histological structure of embryonic tissue. Hence he thought it very probable the true growth had not been examined by him, but only a portion of the surrounding inflamed tissue. This opinion was subsequently confirmed by his examination of a section from a later scraping, which led him to consider the growth to be of an undoubted epitheliomatous nature.

Dr. Cohen said the patient had consulted him in December, 1878, as to the cause of a hoarseness of permanent character, and at that time laryngoscopic examination revealed a small supra-glottic growth, apparently papillomatous, projecting from the ventricle of the right side, about the size of an ordinary white bean, and quite accessible to forceps, by means of which he felt certain that it could have been easily removed. He did not again see the patient until requested by Dr. Seiler to assist him in the operation of tracheotomy, some six months subsequently; during the interval he understood that the patient had been subjected to local applications of astringents and caustics by other practitioners, and he considered this poking at the growth an influential factor not only in causing the growth to increase in size, but probably in changing its clinical significance from benign to malignant, and in producing the abscess of the larynx. The rapid increase in the bulk of the intra-laryngeal neoplasm after partial removal, laryngoscopically, subsequent to the operation, had led him to the belief that the neoplasm was a malignant one with papillary outgrowths; but the usual cachectic clinical manifestations of malignancy had not at any period been apparent. Though he had seen epitheliomas as extensive as the growth in question, and even more so, he had never seen or even read or heard, as far as he could remember at the moment, of a papillomatous formation of this magnitude in the same locality. Dr. Cohen's impression was that the irritation to which the intra-laryngeal growth had been subjected had produced an abscess of the larynx, which had ruptured through the crico-thyroid membrane, which was certainly found open at the second operation to remove the peri-laryngeal growth, and that the peri-laryngeal proliferation had been an extension of the endo-laryngeal growth through the fistula. As the absence of ulceration at any time and of involvement of the skin was unlike the clinical manifestations of undoubted epithelioma of the larynx and thyroid gland, he would ask whether this might not be an example of cylindrical or tubular papilloma or diffused papilloma originating in the ducts of the tubular glands of the laryngeal mucous membrane, and thus presenting the aspects of epithelioma without actual involvement

of the deeper submucous structures. The propriety of excising the larynx had been entertained at one time by some of the gentlemen interested in the case, but had been strenuously opposed by himself, with, as he believed, fully as great a prolongation of life as would have been likely to follow the excision. The tube used at Dr. Cohen's suggestion, as mentioned by the reporter, was an ordinary outer tracheotomy-tube of the quadrant form, but with the segment of the circle continued so as to present downwards instead of forwards as in the usual form of tube, the flange being exceedingly narrow so as to interfere as little as possible with the manipulations. The great advantage of the tube was that the mucus and other matters which might gain access to the tube, whether from the mouth, the trachea, or the lungs, would be coughed out in a direction protecting the face of the operator and assistants from the spattering to which they are not infrequently subjected when the ordinary form of tube is used during manipulations or operation.

Dr. Shakespeare said that since he had unexpectedly heard quoted a so-called opinion of his concerning the nature of the laryngeal growth in question, based upon an examination of the earlier scrapings, he reluctantly arose to take some part in the discussion. He felt impelled to disclaim responsibility for the opinion attributed to him. He had been called upon one morning, during office hours, by his friend the reporter, to look at a section purporting to be made from the scrapings of a growth in the larynx. Patients were in the anteroom awaiting consultation; the reporter himself was also in a hurry, and perhaps five minutes elapsed between the time of his arrival and that of his departure. Within this brief period the history of the case was related, the microscope was brought out and arranged, and the section was looked at. Meanwhile, nothing was said of a request of consulting physicians and surgeons for a careful examination and an opinion, which Dr. Shakespeare several days afterwards accidentally learned had been desired, for the purpose of furnishing information which might influence a decision as to whether the grave operation of extirpation of the larynx would or would not be called for.

It was under these circumstances that a mere *hypothesis* concerning the nature of the growth was arrived at, which the reporter, in his paper written for publication, has dignified with the name of "an opinion." Moreover, this so-called "opinion" had been misquoted, no doubt unintentionally, for, even in this hurriedly-formed hypothesis, the impossibility of absolutely diagnosing a morbid growth by an

examination of the tissue torn from its granulating surface was both recognized and distinctly expressed. The section thus glanced at unquestionably presented the appearances of granulation-tissue covered by an epithelial surface, and the conjecture then formed and expressed was that the section might have been from a sarcoma with secondary papillary formations upon the mucous surface, or from a papilloma experiencing inflammation, or from almost any other morbid growth with its surface inflamed and covered by granulation-tissue.

He declined to be placed in the position of practically advocating the antiquated assumption that the nature of a morbid growth can be recognized, with any great degree of satisfaction or certainty, from an examination solely of scrapings from the surface. There is nothing more delusive and misleading than conclusions based upon such examinations, even when time is consumed in a careful study.

Dr. Shakespeare could not but deprecate a practice which makes one, often unwillingly, a party to the diffusion of such so-called "opinions" as are arrived at in the careless manner already mentioned. He thought that the injudicious publication of just this class of opinions did far more than any other one thing could possibly do to bring discredit upon the microscope, and to block the way of progress in the science of pathological histology. It was in the highest degree unfair to the person whose so-called "opinion" is cited; it could not fail to be alike dangerous to the suffering patient, and delusive to the consulting physician; it could give little satisfaction to the earnest inquirer, whose chief object should always be the laudable search for truth; and it could only add to the confusion of him who would undertake the labor of drawing from the abundance of published observations the general laws which govern disease.

Speaking of the nature of the growth under consideration, Dr. Shakespeare said that he believed, as all other modern students of pathology believed, that it was utterly impossible to make an absolute diagnosis, by means of the microscope, of the nature of a growth upon a mucous membrane without including in the examination a study of the tissue which forms the base of the neoplasm. He had already spoken of the appearances of the sections from the earlier scrapings. The section placed under the microscope for the examination of the members of the Society, and which he understood to be made from the later scrapings, presented to his eye a perfectly typical picture of epithelioma, with numerous cell-nests or pearly bodies scattered here and there in the abundant epithelial plugs which in the main occupy the

field of the microscope. The channels and holes sometimes found in these epithelial pegs, and in many cases filled with granular debris, which Dr. Seiler has singularly described as ducts of glands, he could not recognize as anything else than the cavities which are often found in epithelial accumulations, and which are usually formed by a mucous vacuolation and destruction of a few or a large number of epithelia.

Ulceration and a foul odor in epitheliomatous formations unquestionably are frequent and often early complications. Yet their absence by no means excludes a diagnosis of epithelioma. On the other hand, the presence of epithelial pegs containing cell-nests or pearly bodies can no longer be regarded as absolutely characteristic of an epithelioma, for these onion-like collections of squamous epithelia are not infrequently found in purely irritative superficial accumulations of epithelial cells in the various locations where these cells naturally exist.

This case, therefore, affords one more example of the impossibility of making a reliable microscopic diagnosis of a growth without an examination of the matrix whence it develops.

*December* 11*th*, 1879.

*Report of the Committee on Morbid Growths:*

"A microscopical examination of a thin section made from a portion of the growth removed by scraping the larynx by Dr. Seiler, shows it to be composed of solid anastomosing cylinders or peg-like processes of large epithelial cells; the cells forming the margins of these cylinders are arranged in a very regular manner, and present, in those cylinders which have been cut longitudinally, a somewhat columnar or cubical shape, while in the centre of the cylinders the cells have become larger, more flattened, and their protoplasm has increased in amount, while their nucleus is found to be diminished in size. Upon some of the cylinders the cells are seen arranged in a concentric manner, forming the so-called cell-nests or pearly bodies. Separating the peg-like or cylindrical collections of epithelial cells is observed a fibrillar connective tissue, which presents the appearance of this form of tissue in the various stages of irritation or proliferation, from purely embryonic tissue, consisting of small, round, lymphoid cells without any appreciable intercellular substance, to fully-developed fibrous tissue. From such a histological structure and disposition of elements, your committee are of the opinion that the neoplasm is of an epitheliomatous nature, and may be considered a *lobulated pavement-celled epithelioma*. But

it is to be remembered, however, that the examination is made from a scraping of the surface of a growth, and all such examinations cannot be considered either satisfactory or definite. *January 8th*, 1880."

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2. *Inflammation of the lymphatics of the pulmonary pleura.*

Presented by Dr. E. T. Bruen.

The specimens which I present this evening on inspection apparently reduplicate a similar pair of lungs which I think I exhibited to the Society within the past year. In the former specimen an identical appearance was manifest on the surface of the pulmonary pleura, but there were absolutely no traces of a pathological process in the parenchyma of the lungs or any other organ, which separates the case from the specimens before us.

As I exhibit the left lung, the appearance on the surface of the pulmonary pleura suggests at once the macroscopic appearance of tubercle. Everywhere are seen nodules of a whitish hue as large as a hemp-seed to a small pea; these nodules are connected into a network over the pulmonary pleura by streaks of whitish material evidently inflammatory in their nature. At the base of the right lung there is a pleurisy, much cheesy material, adhesions, and in the lung-tissue patches of chronic catarrhal consolidation. In the left lung, the one covered with the nodules, a few masses the size of a pea are found.

The liver, spleen, kidneys, and mesentery were examined without any pathological appearances being discovered.

These facts discountenance somewhat the idea of tubercle, for such an extensive surface deposit as is seen in this specimen would usually be accompanied by a tubercular deposit distributed throughout at least the pulmonary parenchyma in its bloodvessels, lymphatics, or peribronchial sheaths.

In the specimen presenting a similar appearance on the pleura, to which I have referred, the nodules were examined by Dr. Formad and myself, and no true tubercular structure could be found, but merely evidences of inflammatory cell-proliferation.

It was, then, a simple inflammation of the lymphatics coursing over the surface of the lung in the pulmonary pleura. From the description, I think similar cases were described before the London Pathological Society in their late discussions on tubercle.

The lymphangitis in both my cases, then, developed through the

medium of absorption from the cheesy material in the pleura, the irritant producing tubercle in the last case, perhaps, on account of the diathesis of the patient predisposing to this form of inflammation, as Rindfleisch suggests; and this must be granted if microscopic examination by the Committee on Morbid Growths decides it to be tubercle. But in the former case the lymphangitis was simple in its character, no tubercular structure being detected, unless, indeed, tubercle be simply an inflammation of the lymphatics, in which case the tissue may have represented a very early stage of the inflammation.

*Report of the Committee on Morbid Growths.*—"A section of the lung presented by Dr. Bruen, when examined with the microscope, showed it to have scattered throughout its parenchyma numerous small nodules, which are composed of adenoid tissue. The pathological condition is one of miliary tuberculosis. *March 25th*, 1880."

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3. *Warty growth pendent from uvula; no symptoms.*

Presented by Dr. S. D. Risley.

Mr. C. F. M., æt. 32, consulted me on March 4, 1880, for disease of his left ear. In the attempt to study the condition of the pharynx and orifice of the Eustachian tubes, the uvula was found greatly elongated and its extremity hidden from view below the base of the tongue. It was brought into view by a blunt hook, and a large papillomatous growth discovered pendent to its extremity, the uvula itself being drawn out to fully $3\frac{1}{2}$ cm. in length, and correspondingly diminished in thickness. The growth itself was of a grayish-white color, and appeared macerated; it was 1 cm. in its broadest horizontal diameter, and 8 mm. in the vertical diameter. It presented the characteristic papillary divisions, which were clustered around the end of the uvula like the cauliflower around its stock, presenting posteriorly a hilus down which ran the greatly-distended bloodvessels. The bleeding after excision was very profuse. The accompanying sketch was made on wood by Dr. Lee, after the growth had been in dilute alcohol for several weeks. I at first supposed this specimen to be unique, but my attention was subsequently called to a very similar growth reported and figured *in situ* by Lennox Browne (*The Throat and its Diseases*, Philadelphia, 1878, Henry C. Lea). In his case very distressing symptoms were occasioned. In mine the patient was entirely ignorant of its presence, and had

Warty growth of uvula.

experienced no inconvenience from it—not even a cough. He had a deep, hoarse voice, which very noticeably improved in tone during the two weeks he was under observation.

*March 25th*, 1880.

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### 4. *Secondary sarcoma of the lungs.*

Presented by Dr. H. F. Formad for Dr. R. E. Girvin.

John C., Jr., Philadelphian, age 36 years, laborer in Water Department. He served with the three-months men ; after his discharge he re-enlisted for three years' service in the army. He suffered while there with what was pronounced rheumatism, especially affecting his left leg and ankle. Some eight months after his discharge the ankle troubled him greatly. He applied to a *veterinary surgeon*, who gave him a liniment, which by constant application relieved him for some five years.

In January, 1870, one inch below the internal malleolus of the left foot, a small pimple formed, which caused great pain, of a stinging and throbbing nature. He consulted a physician, who pronounced it a boil. Some weeks afterwards, not being satisfied, physician No. 2 was consulted ; it was pronounced a tumor by him, but at the same time he requested the patient to go to Professor Gross, who diagnosed it a *nerve-tumor*. Three months later, physician No. 2 saw the man ; he operated, and removed a small tumor about the size of a *hazel-nut*, which, upon examination, was found to have encased "a small, detached piece of bone." He was only temporarily relieved. Some six weeks subsequent to this the pain was just as severe as prior to the operation.

Eight months after this a second operation was performed, and a tumor the size of a minié-ball removed.

Even after this operation the tumor continued growing, and after two years it had reached the size of a *plum*, when operation No. 3 was performed for its removal.

During the years 1874 and 1875 the patient tried almost everything in the guise of a remedy. He at length gravitated into the hands of Indian doctors, quacks, etc.

On Easter Monday of 1875 Dr. Leaman examined the foot. Near the 1st of the following December he operated, and the tumor was pro-

nounced "malignant sarcoma." The mass removed was the size of a *goose-egg*. The doctor scraped the bone thoroughly. The wound healed promptly, and he returned to work.

Three years from this date the tumor had again returned. It now was the size of a goose-egg, with another mass somewhat larger surrounding the ankle. Some time during 1878 he fell in with an electrician, who applied his battery to good effect, for after some time the mass softened and ruptured; from time to time he removed portions of the tumor, followed by profuse hemorrhage. This was in December, 1878. Hemorrhages continued, and he became greatly emaciated.

In the early part of 1879 he was taken to the University Hospital. Professor Agnew there amputated the foot and lower third of the leg. Recovery was prompt, and he regained in flesh, after a six-weeks' stay in the hospital, about fifty pounds.

Three months after leaving the hospital he complained of a sense of uneasiness in the breast. In August, 1879, while eating dinner, his mouth filled with blood. Each morning, for some subsequent days, he raised a little blood; through the night he raised small tough masses the size of a pea, slightly tinged with blood. This continued until he had a profuse hemorrhage. Now he consulted an experienced practitioner, who attended him during these attacks of hæmoptysis, which occurred at first twice daily, and afterwards every third day.

Preceding these attacks he would have a violent fit of coughing, which lasted until he coughed up a mass having a *tubular appearance*, resembling a solid quill, varying in size from one inch to three inches in length. He could tell almost to the hour when a hemorrhage was coming, and could detect these masses moving in the "bronchial tubes" until they were broken off and coughed out, followed by hemorrhage. From time to time hemorrhagic spots of a *dark-purple* color made their appearance upon the skin; these were quite painful to the touch; they disappeared in twenty-four hours.

At this time a consultation was held by the physician in charge. After consultation and microscopic examination of one of the masses, it was pronounced "diphtheritic bronchitis," and treated accordingly. These periodic attacks of hæmoptysis greatly reduced the patient in weight and strength.

In November, 1879, I first became acquainted with the case. There was dulness, unaltered by position; weakened or absent breath-sounds over the greater portion of the lungs. The right lung was apparently useless, the left side of the chest only seeming to expand and do the

work of carrying on respiration. Great dyspnœa and constant cough, with very little expectoration; night-sweats; the skin normal in appearance; no *cancerous tint.* Small masses were frequently found in the expectorated sputa. Twice such little masses had been sent to Dr. Formad without history, and were pronounced in his report "fragments of an organized new growth, evidently round-celled sarcoma."

This formed the basis for my diagnosis.

Patient died April 26, 1880.

*Autopsy* was held in the presence of Dr. Formad. The lungs were found, upon exposure to view, to fill the thorax thoroughly. The surface of the lungs, as well as the parenchyma, was studded with numerous nodular masses of new growth, varying in size from one to six inches in diameter.

The macroscopical appearance of the lungs is very accurately shown in the accompanying drawing made by Dr. W. F. Lee:—

Sarcoma of Lungs.

The intercostal pleura was perfectly free from any deposit whatever; no pleuritic adhesions. The kidneys and liver were nornal, only greatly congested. There were no enlargements of the mesenteric glands.

Microscopic examination of the new growth of the lung made by Dr. H. F. Formad showed it to be a small round-celled sarcoma of very typical structure. *June 24th*, 1880.

5. *Case of phthisis and tuberculosis—Enormous cavity—Physical signs of pneumothorax.*

Presented by Dr. J. H. Musser.

W. M., æt. 25, admitted to University Hospital September 13, 1880; native and resident of Philadelphia; ornamental japanner; spree occasionally; used considerable tobacco; had gonorrhœa; thinks his "break-down" due to venereal excess in youth; single, a brother living; two sisters died when children; mother living; all his brothers and sisters died of consumption; history of father's family not known, always healthy; present troubles date back fifteen months; subject always to a slight winter cough. In July, 1879, was overheated; cooled suddenly, causing a chill, followed by a pain in the head and breast, and a cough with frothy expectoration; no blood. In two weeks, on account of cough, pain in the left lung, and debility, had to quit work. Soon resumed work, and in a short time lost appetite; had fever and night-sweats; cough grew worse, expectoration more purulent; became weaker and weaker; worked all winter, but in March had to give up; never went to bed until he entered hospital. All spring was very weak; throughout summer, with the cough and hectic, had diarrhœa; two or three passages a day, without pain, not fetid, sometimes dark, again green; late in summer indigestion and vomiting ensued.

When admitted, was very feeble and very much emaciated; pale, anæmic face, brown hair, blue eyes, lashes not long; pearly sclerotics and pale conjunctivæ; countenance of anguish; skin moist, fingers slightly clubbed; fever and night-sweats. Appetite poor, capricious; tongue slightly coated all over, not flabby; weight and fulness after meals, flatulence, poor digestion; bowels constipated; hemorrhoids; at intervals slight abdominal pains; liver extends one inch below ribs; spleen not enlarged. Very slight expansion of left lung; vocal fremitus absent anteriorly and laterally; at base slightly increased; anteriorly and posteriorly amphoric voice, at the base increased vocal resonance; on percussion, upper lobe tympanitic; dulness over the lower lobe, in front, at the side, and behind; on auscultation of upper lobe, pure amphoric breathing, respiratory murmur absent; a few crackling râles on inspiration heard over lower lobe. Right lung at apex and base expanding very much; vocal fremitus normal at apex and lessened at base; vocal resonance the same. Vesiculo-tympanitic percussion-note.

At apex, front and back, prolonged expiration, harsh respiration, and a few mucous râles; puerile respiration over remainder of lung. Apex of heart in sixth interspace, one and one-half inches inside of nipple-line; impulse feeble; valvular element of first sound distinct; pulse rapid, feeble, compressible; urine normal.

From date of above note to October 17, strength failed more and more. The physical signs continued about the same. At times there were more râles at the right apex than at other times. The chest-pains increased, the right abdominal pains continued, the cough became more harassing, the hectic increased, the pulse was very rapid. On the 17th he complained of pain in the right side, and apparently friction râles were heard; the expectoration was streaked with blood. The morning of the 20th hemorrhage began, continuing an hour. During that time a pint and a half of bright arterial blood was raised, in mouthfuls, without effort. On auscultation, large bubbling râles were heard at the base of the lung; amphoric breathing and metallic râles over the cavity; very weak, pulse 134, hurried shallow breathing.

*October* 20. Since last note, has been expectorating currant-jelly clots at intervals; cough very severe; diarrhœa; feels easier. At 11.30 A. M., after above note, suddenly, without cause, profuse discharge of blood from mouth and nose; death in about five minutes.

*Post-mortem examination* four hours after death, by Dr. W. E. Hughes.

Skin yellowish. Epidermis scaling off in large flakes on the abdomen. Body much emaciated. No *rigor mortis*. Nostrils and mouth contained clots of blood.

On section, tissues dry and pale, very little blood exuding from the cut surface. Scarcely any fat. Muscles pale red.

*Thorax.*—Pericardium contained half an ounce of straw-colored fluid. Heart normal size, muscle not fatty to the naked eye. Right ventricle and auricle contained a chicken-fat clot. All the valves stood the hydrostatic test. The aortic valves were thickened slightly, as were also the mitral leaflets. In the latter were a few small calcareous deposits.

At the apex of the right lung a few pleural adhesions. Entire visceral and parietal layers of the left pleura firmly united and thickened, save in the lateral region. In this area, over the lower lobe, a sac was formed an inch deep, three inches wide, and four inches longitudinally, containing a few ounces of fluid with some lymph. The upper

lobe of the right lung was partially solidified. On section, two small cavities, the largest an inch in diameter, both filled with a gray grumous fluid, occupied the centre of the solidification. The consolidated portion was of a gray color, with numerous small cheesy points in it. In the lower lobe there were numerous small masses of tubercle. Remainder of lung emphysematous. The vesicular tissue of the upper lobe of the left lung was entirely destroyed. The walls of the cavity were formed of the thickened pleura, while across it extended thickened cords and bands containing the remains of obliterated vessels and bronchi. Numerous stumps projected from the sides of the cavity, and on its walls were studded small cheesy masses. The cavity was filled with blood and some puriform matter. The lower lobe was consolidated. Numerous cheesy masses and a few cavities were found in the gray consolidation. The bleeding-point of the hemorrhage was in one of these cavities. The edges of the lower lobe were lobulated, apparently congenital.

*Abdominal cavity.*—It contained a pint and a half of straw-colored fluid. Intestines presented externally, at varying intervals, dark-red blotches. Liver weighed four and a half pounds; pale and fatty, slightly cirrhotic at lower edges. Gall-bladder contained a little bile. Spleen weighed twelve ounces; was soft; dark red in color. Kidneys weighed ten ounces each. They were fatty; capsule not adherent. Stomach at largest part only one and a half inches in diameter. Rugæ very prominent. Mucous membrane very pale. A small circular ulcer with raised indurated edges in the duodenum, four inches from the pylorus. Two similar ulcers in the jejunum. All the upper Peyer's patches ulcerated, only very slightly, as if just beginning; some of the patches had two or three transverse excavations. The lower patches were elevated and rough, as if tubercle had just developed. In the cæcum numerous small ulcers similar to those in the small intestine. Ulcers less numerous in the ascending and absent in the transverse colon. Mesenteric glands not enlarged.

Brain healthy. Slight increase of arachnoid fluid.

On account of the physical signs presented during life, the mode of death, and the extensive cavity found after death, the case is of interest. The physical signs of consolidation and cavity of the right lung were masked by the compensatory emphysema. The signs of the cavity of the left lung were so like those of pneumothorax that its occurrence was considered.

Death from hemorrhage is rare in phthisis. It is of interest to note that the bleeding did not take place in the large cavity, but from a vessel in one of the small cavities in the lower lobe.

*December* 23*d*, 1880.

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6. *Removal of left superior maxilla, left malar, nasal, lachrymal, lateral mass of ethmoid and pterygoid processes of sphenoid bone for tubular epitheliomatous pharyngeal polypus.*

Presented by Dr. C. B. Nancrede.

Charles Muscavitz, aged 52 years, a Pole, was admitted into the male surgical ward of the Protestant Episcopal Hospital in the early part of September, 1880. He most positively asserted that he had noticed nothing until two months before coming to me, when his eye began to be protruded. When I first saw him his left eye was displaced outwards and upwards by a firm, hard mass, seemingly involving the internal angular process of the frontal bone, the nasal process of the superior maxillary, and the orbital plate of the same bone to a limited extent. There was no bulging of the infraorbital region, but, the finger being introduced into the mouth, a slight fulness with tenderness could be detected beneath the zygoma. The posterior portion of the hard palate was slightly depressed, while the soft palate was bulged downwards. By carrying the finger behind the soft palate into the pharynx a large, irregular, lobulated mass was detected, completely filling up this cavity, except perhaps towards its right moiety. The nasal process of the left superior maxilla and the nasal bones were pushed upwards and outwards by the extension of the growth into the left nasal chamber. Indeed, polypoid masses could be detected by both the eye and finger, completely filling the left nostril. I diagnosed a malignant pharyngeal polypus. After consultation with my colleagues, I determined to extirpate the growth with the involved bones. The patient having been first etherized, the Fergusson incision was made and the flap reflected. All hemorrhage having been arrested, chloroform was administered in the place of ether, and the operation completed in the ordinary manner, with a few exceptions presently to be mentioned. Owing to the failure of the bone-forceps to divide the zygoma, the whole malar bone came away, from some lateral motion in using the instrument. Only a part of the tumor came away with the superior

maxilla, owing to its firm connections with the roof of the pharynx. The portions of tumor left after the removal of the upper jaw were torn away by the fingers or Fergusson forceps, or cut away with scissors. Owing to the able manner in which I was assisted by my colleagues, Drs. Forbes and Hunter, very little blood was lost. One unusual circumstance was the ease with which hemorrhage was arrested, owing to the fortunate circumstance of the stump of the internal maxillary artery projecting some half inch or more, thus admitting of its ligation. Only a very sparing use of the hot iron was requisite. The whole wound was wiped out with a solution of chloride of zinc. The case did well, with the exception of sloughing of the cornea, some two weeks after the operation, from secondary implication of the nerve, either by inflammation or extension, perhaps, of disease into the sphenoidal fissure. Examination showed that the whole left superior maxilla, the left lateral mass of the ethmoid, part of the vertical plate of the same bone, the left nasal, the lachrymal, the left malar, and both left pterygoid processes of the sphenoid had been removed.

*Microscopical Report, by Dr. J. H. C. Simes.*—"The growth, upon microscopic examination, is seen to consist of epithelial cells forming solid rods or peg-like prolongations, which branch and anastomose with other similar prolongations. Between the tubes is a small amount of fibrous tissue infiltrated with embryonic cells. In some of the pegs the cells have arranged themselves in a concentric manner, forming, somewhat imperfectly, cell-nests or pearly bodies. This histological arrangement of elements would place the growth under the epitheliomata, and, according to Cornil and Ranvier, with the tubular variety of this class of tumors." *October 28th*, 1880.

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### 7. *Acute caseous phthisis.*

Exhibited by Dr. J. H. Musser.

When removed from the body, the lungs which are before you this evening presented the following appearances. *Right* lung: costal and pulmonary pleura adherent; entire lung consolidated. On section, upper lobe of a yellow color, very soft, cheesy, presenting numerous small cavities, from which poured a thick gray fluid. A strong stream of water playing on the lobe washed out the soft, cheesy material and revealed the lung riddled with cavities not larger than a filbert, gene-

rally communicating. The middle lobe was completely consolidated, of a gray color, very slightly granular, and exuded a thin fluid. The lower lobe was grayish-yellow, soft, with three small cavities. *Left* lung: pleural adhesions at apex; one-fourth of the upper lobe presented the characteristic appearances of catarrhal pneumonia. The remaining organs were normal.

The specimens were removed from a young man 25 years of age; single; a painter; of good habits; without any hereditary predisposition to disease. He had always enjoyed good health, and had never been subject to cold. One month prior to admission to the hospital he caught "cold." Persistent cough was the only symptom for two weeks. Then he noticed feverishness and night-sweats. The cough, at first dry, about the same time became muco-purulent. Emaciation and loss of strength followed. His appetite remained very good, and there was no gastro-intestinal derangement. During this time and until admission to the hospital he was going about from place to place in the Eastern States in search of work. The past week the fever and sweats and the cough have continued, the emaciation has progressed, and the debility has become greater.

On admission, November 5th, haggard and emaciated; dry hot skin throughout the day; exhaustive night-sweats; face pale; hectic flush; eyes blue; sclerotics pearly; hair brown; lips thin and pale; hands bluish; fingers not clubbed. Special senses and cerebral faculties normal. Teeth poor; tongue red, clean; appetite and digestion very good; no diarrhœa; liver and spleen normal. Pulse 110; heart-sounds normal, respirations 28; almost constant cough, worse at night; abundant muco-purulent expectoration; great pain in right side and at right apex, with tenderness; unable to lie on that side. Physical signs of consolidation of right upper lobe, anteriorly and posteriorly, with mucous râles.

*November* 7. The severe cough has continued; extreme pain; fever and profuse sweats; emaciation progressing; weak. The dulness, the bronchial breathing, and bronchophony of the right apex have been replaced by cracked-pot sound, amphoric breathing, and pectoriloquy. These signs are especially distinct between the clavicle and the third rib. Complete consolidation of the middle lobe is noted, and partial consolidation of the lower.

21*st*. The physical signs of a cavity noted last week continue, but are variable. At one time cracked-pot sound is absent and tympany present, cavernous breathing replacing the amphoric. With

amphoric breathing there are a few large râles; when cavernous, innumerable moist râles are heard. The more distinct evidences of consolidation of the lower lobe were frequently pronounced with dulness, bronchial breathing, and subcrepitant râles. No alteration of digestion; pain and cough only relieved by morphia; fever and sweats continued; breathing hurried.

21*st*. The distinct but variable pectoriloquy and other signs of a cavity continue. At the base complete consolidation and softening; large mucous râles heard all over the chest. Impaired resonance and prolonged expiration, with a few crackling râles, noted three days ago at left apex; they continue.

On November 30th the following physical signs were noted. Thorax long and flat; considerable flattening above and below right clavicle; expansion impaired; vocal fremitus increased over lung anteriorly, diminished at base posteriorly. On light percussion, in first interspace at right edge of sternum, tympany becoming dull towards shoulder; on deep percussion, cracked-pot sound. In second interspace, on light percussion, tympany especially on full inspiration; at end of expiration, dulness; on deep percussion, cracked-pot sound. From the third rib to the liver, vesiculo-tympanitic percussion-note. On auscultation, cavernous breathing of high pitch, becoming amphoric towards the shoulder, with large bubbling râles in the first interspace. In second interspace, cavernous breathing and bubbling râles; from third rib, cavernous breathing and mucous râles. Pectoriloquy absent to-day, though at times distinct in the three upper interspaces; vocal resonance absent. In line of axilla, dulness from above downwards. Posteriorly, dulness to middle of scapula, increased resonance below. Where dulness, vocal fremitus increased, absent where extra-resonant. Above spine of scapula, amphoric breathing and bubbling râles; from spine to middle of scapula, blowing, almost cavernous breathing in inspiration, while only moist crackling râles were heard in expiration. Below middle of scapula numerous moist râles and a very faint blowing breathing. Left apex as previously noted.

Hectic very great; delirium at night; muco-purulent expectoration, slightly streaked with blood; daily epistaxis; tongue clean and red; bowels costive; appetite good.

*December* 6. Cracked-pot sound from clavicle to fifth rib on right side, in first interspace on left; other signs the same; continued low delirium and stupor.

16*th*. No noteworthy change in physical signs. Died of exhaustion to-day.

Note first the pathological conditions of the lungs. Upper lobe, numerous cavities; middle lobe, consolidation; lower lobe, consolidation, caseation, a few cavities. Note that over the upper lobe anteriorly there was tympany or cracked-pot sound, cavernous or amphoric breathing, variable pectoriloquy; posteriorly, dulness and amphoric breathing; physical condition, innumerable small cavities. Over middle lobe, vesico-tympanitic note and cavernous breathing in front, dulness and blowing breathing behind; lobe consolidated. The lower lobe completely consolidated, with a few cavities yielding vesiculo-tympanitic percussion-note all over; cavernous breathing anteriorly, faintly blowing posteriorly. The intermittency of the pectoriloquy and the variability of the auscultatory and percussion phenomena are of interest.

There was no mistake in the above signs, as they were demonstrated to ward classes, and from time to time compared with the physical signs given by the enormous cavity in the lungs which I presented December 13th. I neglected to note, also, that the cavernous or amphoric breathing was often heard more clearly in inspiration.

*January* 27*th*, 1881.

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8. *Carcinomatous tumor of the mediastinal glands and of the lung.*

Presented by Dr. Edward T. Bruen.

I cannot present this specimen with the clinical study which its importance demands, because the patient died the day after I resumed charge of my wards in Blockley, and was almost moribund when I saw him. The immediate cause of death seemed to be dyspnœa from the presence of a large right-sided pleural effusion. Tapping of the chest was accomplished, and three pints of liquid were withdrawn, but the patient died a few hours afterwards.

*Post-mortem*.—The body was fairly nourished. Rigor mortis marked. On removing the sternum, the large mass now exhibited was found filling the entire mediastinum and involving the cervical glands. The cervical glands presented a mass as large as a small bunch of grapes, reaching up some distance above the clavicles on both sides. No glandular enlargements were noted elsewhere. The costal

pleura was healthy, but many nodules were scattered through the right lung and pulmonary pleura. Elsewhere all the tissues and organs were notably normal. I think during life the dulness over the mediastinum, contrasted with the flatness over the effusion, and the presence of the enlarged glands in the neck, would have been helpful aids to establish the diagnosis. One of the earliest symptoms in these cases has been pain, sometimes localized, sometimes radiating down the chest or down the arms. This is a really fundamental symptom, and is useful before percussion can be employed. The difficulty confronted in some cases is to distinguish this pain from that caused by commencing aneurism before the aneurism is attested by a murmur. This suggestion applies especially to that form of aneurism known as dilatation of the aorta. The effusion must have taken place in consequence of inflammation of the pleura, since many shreds of lymph were found floating in the fluid; but the pressure from these enlarged glands might have produced the same effect. I have under observation a case of adenia, or enlargement of the bronchial and cervical glands, without evidence of either corpuscular blood-change or scrofula or syphilis, occurring in a young man about thirty years of age. In this case, although the interference with the intrathoracic circulation has caused a general enlargement of the superficial veins of the body, there is no pleural effusion. I think that the pleural effusions in cases of mediastinal cancer mean oftenest a secondary cancer of the pleuræ. A microscopic examination of this growth showed its structure to be that of a carcinoma of the medullary variety. A section prepared by Dr. Formad is ready for your inspection. *Nov.* 11*th*, 1881.

## V. THE GENITO-URINARY APPARATUS.

### *a.* KIDNEYS AND BLADDER.

#### 1. *Medullary carcinoma of the bladder.*

Presented by Dr. Joseph Hearn.

Francis B., æt. 59, married, a blacksmith, was treated at the surgical out-patient department of the Jefferson Medical College Hospital during the past summer for vesical trouble, under which he had labored for twelve years. The earliest symptoms were frequent micturition and slight pain at the completion of the act: they gradually grew worse, until three years ago, when he passed blood for the first time. Of late the discharge of blood was increased by unusual exercise, the use of malt or spirituous liquors, and by sexual indulgence; at times it was quite profuse, and most marked after the expulsion of the urine. Two months before the fatal issue he was compelled to take to his bed from the loss of strength and flesh, and on account of constant pain, which was at times intense, referred to the pelvic region and to the glans penis, the latter of which he was constantly pulling at and squeezing, even when he was unconscious. On the introduction of a sound the surface of the bladder was felt to be roughened and uneven. The employment of the instrument was productive of great pain, and occasioned a copious hemorrhage. Unless under the influence of the anodynes, he passed a few drops of urine every thirty minutes during the day and night. He was partly unconscious for two weeks before death, and for one week suffered from incontinence of urine.

At the post-mortem examination, made September 11, 1879, twenty-four hours after death, on incising the linea alba, a rather firm, grayish-white nodule, as large as a hazel-nut, and seated in the parietal peritoneum, was cut through about midway between the umbilicus and pubes. With the hand the peritoneum was found to be the seat of other secondary growths, but in deference to the wishes of the family the incisions were restricted to making an opening sufficiently large to remove the bladder. The capacity of the viscus was not diminished.

The base was thickened to the extent of nearly one inch by a soft encephaloid mass, which protruded several small nodules into the space between the seminal vesicles. On opening the bladder and emptying it of its muco-purulent urine, the entire mucous membrane was seen to be corrugated and inflamed. The *bas-fond* and the greater portion of the organ were converted into irregular fungous and ulcerated masses, which infiltrated all of its coats except the peritoneal. On section they were of a pinkish-white tint, soft, and very friable. The prostate was only slightly enlarged, and was not invaded by the disease. Microscopical examination by Dr. S. W. Gross showed that the primary growth of the bladder and the secondary nodule of the peritoneum were composed of large polymorphous epithelial cells, the majority of which had undergone fatty changes, contained in alveolar spaces formed either by muscular fibres, or by fibrous tissue.

Dr. S. W. Gross remarked that primary carcinoma of the bladder is to be ranked among the rarest of affections, although it is not very uncommon as the result of the extension of a similar growth from the contiguous and associated organs. The case narrated by Dr. Hearn is of interest on account of the long duration of the disease, death usually ensuing in less than twelve months. Examples of the prolongation of life for eight or nine years have, however, been recorded by Sir Henry Thompson, and W. M. Clarke. They were all villous medullary carcinomas, and hemorrhage was a prominent symptom from the first. *Nov. 13th*, 1879.

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2. *Kidneys, ureters, bladder, and calculi from a case of impacted calculus in a child twice successfully lithotomized — Death from exhaustion.*

Presented by Dr. James Tyson.

The specimens were removed from a lad 6 years and 4 months old. On April 21, 1877, when 3 years and 10 months old, he was cut for stone by Professor D. Hayes Agnew, who removed the phosphatic calculus now exhibited to the Society. It was oval, 3.5 cm. long by 2.2 cm. in its longest conjugate diameter, white, smooth on its surface; on section, also white, loose in the texture of its central portion, and without a nucleus. It was of the same composition throughout. The cut made by the operation was very slow in healing, the latter not being

completed until October 1. At least two years before the operation—that is, when less than two years old—he began to complain of bladder symptoms; previous to this he had been delicate. He came under my care about May 1, 1878, one year after the operation for stone. He was then suffering with the symptoms of cystitis, such as are present with a calculus in the bladder, but usually subside soon after the removal of the stone. There was frequent micturition, a small amount of pus, and a very small degree of albuminuria, while the urine was either alkaline when passed or became so very soon afterwards. He was subject, however, in addition, to paroxysms of extreme pain, which was confined almost exclusively to the region of the navel. The attacks occurred about once a week, and succeeded upon days on which he felt unusually well, played a good deal, and tired himself thereby; they lasted until paregoric sufficient to relieve them had been given. He was very pale, anæmic, and appeared a very delicate child.

I put him on benzoic acid, which was to be given until the urine was distinctly acid, and at the end of two weeks he was greatly better. The paroxysms were less frequent; there was less pus in the urine. To the benzoic acid treatment was added sandal-wood oil, and the two were continued, more or less constantly, either together or alternately, as the condition of the urine and other symptoms suggested, throughout the entire summer. This was spent at the seaside. Iron and quinine were also given as tonics when required. It was considered that during this time there was a gradual abatement of his severer symptoms.

About September 10th he had a decided return of the symptoms, this time accompanied with great pain over the bladder, and frequent micturition, during which he seized his penis in his hands; he passed water from three to six times in a night. His general health I thought at this time greatly improved. The urine was very pale, almost colorless, acid, specific gravity 1003, and contained a few leucocytes and a small quantity of albumen. By the middle of October he was again relieved of the painful symptoms, the specific gravity of the urine increased to 1007, but the albumen had increased decidedly to half the bulk of urine tested. No tube-casts could be found, nor were any ever found in the entire course of the sickness,—at least, while he was under my observation.

By the middle of January, 1879, the albumen had again become very small in quantity, there was a scanty sediment of leucocytes, and the urine had attained a specific gravity of 1016,—the highest it ever

was while he was under my care. The special treatment during this time was sandal-wood oil, directed to the cystitis, and benzoic acid, to influence the reaction of the urine, as required.

On February 3, 1879, he had a hemorrhage from the bladder, accompanied by great pain. It was, however, at a single act of micturition, and what I examined by the microscope, eight hours after this, contained no blood-corpuscles, but one-half its bulk of albumen. The hemorrhage did not recur.

February 12th, Professor Agnew sounded for stone successfully. On March 11th he operated, and after some difficulty removed the second stone exhibited. This was much smaller than the first, being also oval, 2 cm. long by .6 cm. wide. More than half the external surface was white, the remainder brown, and projecting from the former were a number of spicules, by which it was supposed it was imbedded in corresponding depressions in the bladder; whence the difficulty in extracting it. On section, the external concentric laminæ were white, but in the centre was a brown, oval nucleus, 1 cm. long and .5 cm. wide. Portions of this nucleus almost totally disappeared on incineration, but the residue responded to the murexid test. It therefore contained some uric acid.

He was kept in bed six weeks, until April 30, when he was allowed to get up, although the wound was not quite healed. Until April 15 there was a good deal of pus and some blood in the urine, but upon that date we returned to the treatment by sandal-wood oil, after which they rapidly diminished. He had some pain on passing water, which continued until April 15, when this too disappeared. Immediately after getting up, the urine dribbled through the opening in considerable amount, but this gradually diminished until it was scarcely sufficient to soil a napkin during the entire day. On May 10 the urine contained a mere trace of albumen, a small sediment composed of pus and earthy phosphates, was faintly acid when passed, but became alkaline soon thereafter, and presented a specific gravity of 1005. Soon after this he went to the seashore.

On August 14 the little patient's mother called upon me to report his condition. She said that for as much as two weeks at a time there was no dribbling of urine from the wound; then an attack of pain was succeeded by dribbling for a time. He was very well in every other respect, although thin. He ate heartily and played all day long. He continued taking the sandal-wood oil, and for a time benzoic acid.

Early in September, Willie returned from the seaside. He was

very thin, but his wound had entirely healed. His mother reported that soon after his return he took cold two or three times in rapid succession, his stomach became deranged, his appetite disappeared, and he became weak and emaciated. I saw him September 20, the first time since his return, and was much struck by his emaciated and cachectic appearance. The urine presented about the same characters as when last examined. He was immediately put to bed, placed on restorative treatment, and for a time seemed to grow stronger; but, apparently in consequence of the absence of treatment especially directed to the urine, the latter contained much more mucus and became alkaline, and these characters continuing the benzoic acid was re-ordered, when they partially disappeared.

The improvement in his general condition was, however, only temporary, and early in November it was evident he was again declining. He emaciated, grew weaker day by day, and died on the afternoon of November 15, 1879, although he was out in his coach two days before. On the day before his death he had to be catheterized, but there was considerable urine in his bladder, and there was no reason to suppose there was suppression of the secretion. He also suffered a great deal of abdominal pain for two or three days before he died. Very interesting in connection with the results of the post-mortem examination is the fact that his father, while feeling his abdomen on the afternoon before he died, discovered a hard, irregular mass in the neighborhood of the umbilicus.

The *post-mortem examination* was made fifty hours after death. The body was extremely emaciated, but all the organs were found normal except the kidneys, ureters, and bladder. Both kidneys were cystic, each being converted into a multilocular cyst, of which the loculi were distended with a mixture of pus and urine, the former apparently predominating, as there was very little urinous odor. The left kidney was the larger, measuring 10.5 cm. by 5.5 cm. Its surface contained numerous white tubercles (see A, A in the figure), ranging in size from that of a pin's head to a pea. The *pelvis* of this *left kidney* was distended with the same fluid which filled the loculi, and the left ureter—dilated throughout its extent—was almost completely occluded by a single calculus of most peculiar shape. It was spirally twisted and somewhat spindle-shaped, much constricted near its middle, and 5.5 cm. long by 1.5 cm. in its thickest part.

The *right kidney* was smaller, measuring 8.5 cm. by 4 cm., but in all other respects similar. The pelvis and ureter were dilated, and

the latter packed with three fragments of what was originally a single stone, constricted at two points, where it had probably been fractured in removal, making three pieces. The original stone was arcuate in shape, 10.5 cm. long and 1 cm. wide at its middle. The same tubercles were scattered throughout the cortex of this kidney.

The *bladder* contained a small stone, which had evidently descended since the last operation, and was apparently a fragment of one of the stones remaining in the ureter, the fracture having taken place at a point of constriction. This stone was about 2.5 cm. long, and varied in diameter from .5 to .75 cm. The bladder was thickened, the mucous membrane thickened, and no traces of the cut made at the time of the operation were discoverable.

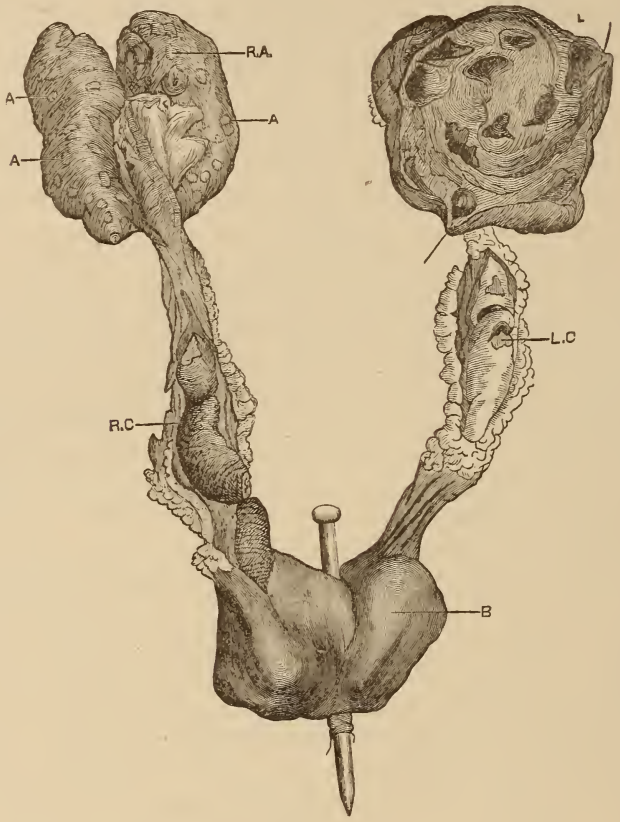
The pedunculation observed on the stone removed at the last operation, which was believed to have occurred during its presence in the bladder, most probably did not occur there, as the stones found in the ureter exhibited the same peculiarities, although these pedicles were probably the means of its attachment to the bladder.

*Remarks.*—The case is a rarely interesting one, not only in its unique morbid anatomy, and as showing the rapidity and extent of sedimentary processes in the urine, but also in suggesting a possible explanation of certain recurrences of stone in the bladder; while from the therapeutic aspect it is not without its uses.

In the first place, it is evident that the hard mass felt by the father of the child through the abdominal walls just before death was made up by the calculi, which, although contained in separate ureters, united to form a single confused mass in the median line just above the sacrum. How long before death this could have been felt I do not know, for I had not examined the abdomen for some time. There is every reason to believe, however, that it might have been recognized at any time subsequent to the emaciation, which began during his absence from the city, in the summer, and which so impressed me when I first saw him, some time after his return. The practical hint to be deduced from it is that frequent examinations of the abdomen should be made in cases of suspected impacted calculus.

A second interesting question refers to the cause of the peculiar spiral, corkscrew twist in the calculus in the left ureter, and the approximation to this appearance in the constrictions in the stone in the right ureter. At first I was inclined to account for this by some peculiarity in the mode of deposit; but could it not have been the result of the solvent action of the urine as it trickled round the stone in seeking an

outlet from the pelves of the kidneys through the ureters, the lumina of which were so tightly filled by the calculi? Support to this possibility is afforded by the fact that the stones are phosphatic, and that




the urine was kept acid more or less constantly by the benzoic acid, and by the fact that there were no concretions in the kidneys themselves and their pelves—only in the lower halves of the ureters.

A third point of interest bearing upon the termination of the case was the relation of the walls of the left ureter to this spiral channelling of the stone contained in it. They were closely applied to it, as the threads of a female screw are to those of the male screw which passes through it. Hence the impaction was complete, and it was impossible for this calculus to descend farther. The impaction of the stone in

the right ureter was almost as complete, and it was impossible to move either until the ureter was slit up, while with great difficulty were the folds reached which applied themselves to the spiral channels.

Fourth, certain recurrences of stone in the bladder receive a lucid explanation by this case. It will be remembered that two stones had been removed at successive operations, and one was found in the bladder at the post-mortem examination. The first removed, judging from its size and shape, was evidently a separate stone; the second small stone removed by operation, although its ends were both rounded off, corresponded in size with one of the fragments of the stone in the right ureter, and the little prominences by which it was imbedded in the walls of the bladder, causing its adherence to the latter and consequent difficult removal, were precisely similar to others on the impacted stones. Hence it was very likely a fragment which had broken off from the parent stone at one of the constricted points, after it had been extruded a certain distance into the bladder; for the stone which was found in the bladder at the post-mortem examination had evidently recently broken off from the parent stone, as it presented a faceted end, which fitted another on the lower end of the impacted stone. And it is not at all impossible that had the child lived another portion could have broken off and been projected into the bladder.

Finally, the therapeutics of the case show indisputably the efficiency, first, of sandal-wood oil in catarrhal states of the bladder, and, second, of benzoic acid in securing an acid reaction in urine which has a tendency to be alkaline. Tannic acid in sufficiently large doses was substituted for the benzoic acid for a time, without the same effect in diminishing the alkalinity of the urine and thereby diminishing the viscidity of the mucus.

With regard to the administration of the benzoic acid, it was given in the shape of compressed pill, which, after a good deal of experience, I am convinced is by far the best mode of administration of this otherwise most unpleasant remedy. The pill thus prepared is tasteless, is swallowed with great facility, does not irritate the stomach, and the remedy is as efficient in this as in any other mode of administration. The dose given was at first half a grain three times a day, which was afterwards increased to five grains, as often, and continued for weeks, only with advantage. It had been discontinued, however, for some weeks previous to the decline of appetite and the gastric derangement, which appeared two months before he died. During the month before his death it was found necessary to return to it, in consequence

of its useful effects upon the urine, which the untiring vigilance of the boy's mother had not failed to recognize.

It is scarcely necessary to say that the sandal-wood oil was found best administered in capsules. In this shape it could never be discovered that it caused any gastric derangement.

*January* 22*d*, 1880.

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### 3. *Cystic kidneys, with renal asthma.*

Presented by Dr. E. T. Bruen.

The kidneys which I exhibit to the Society this evening almost duplicate those which Dr. Tyson exhibited not very long since. They are in a state of advanced cystic degeneration. Of the left kidney hardly a vestige of renal substance exists, merely the capsule of condensed connective tissue and bloodvessels remaining. The right is very nearly as extensively altered, although there is probably more renal tissue. In the saccular spaces I found a large number of stones—some ten or twelve—the size of which ranged from that of a cherry-stone to that of a large walnut. The right kidney weighed six ounces, the left two ounces. The right measured longitudinally five and a quarter inches, transversely two and three-quarter inches. The left measured longitudinally two and a half inches, transversely one and three-quarter inches.

The ureters are much dilated; that from the right kidney is the larger, and would admit a No. 24 English sound. The bladder appeared somewhat thickened, and the openings of the ureters were large enough for me to introduce my finger within them. There were no stones either in the bladder or in the ureters.

The patient's history was that of a healthy man until the age of 60; he died when 65. During the last five years of life he suffered from bronchitis and emphysema. He was under my charge about two weeks, during which time about seventy ounces of urine were passed daily. It was slightly albuminous, sp. gr. 1006, no casts. There was slight anasarca of the lower limbs; this he told me had never been worse than I saw it, and he had never concerned himself about it. From hypertrophy of the heart, accentuated second sound, and other general symptoms, I believed him to be suffering from interstitial nephritis, which was practically the case. The chief object that I have

in exhibiting these specimens is to invite attention to the peculiar symptoms which preceded death. I found him one morning with all the symptoms of asthma—pale face, anxious expression, cold, clammy skin, superficial veins distended by blood, violent respiratory efforts, and much suffering from want of breath. To my surprise, on auscultating the chest, air entered and left it without bronchial or laryngeal obstruction: the heart was acting with sufficient power to carry on the circulation. There were no râles to be heard. The determining cause of this condition I learned to have been an altercation, from which time the patient had grown worse.

I believed the asthma to be uræmic, due to the retention in the blood of those organic materials which it was the function of the kidneys to excrete, but which had not been found in the urine; moreover, I recalled that the skin had always been dry and harsh. The organic materials thus retained had acted as irritants to the terminal filaments of the vaso-motor nerves, producing a spasm which was manifested in the lungs by the deficient aëration of the blood and the dyspnœa. This species of asthma has been described by Dr. Allbutt in the *British Medical Journal* of September, 1877, and alluded to by Dr. Johnson in the same journal in the Lumleian Lectures. That retention of the nitrogenous material is capable of producing increased vaso-motor tension has been proved by the experiments of Dr. Parkes, published in the *Lancet* of May, 1874, in which he states that after having kept a number of soldiers upon a non-nitrogenous diet, and then upon a nitrogenous diet, he found that the arterial pressure was proportionate to the increase or diminution of the nitrogenous food which was supplied in excess of the wants of the economy. If, then, the kidneys or the skin fail in their functional activity, how much the more readily can we understand an exaggerated expression of the evils of an increased condition of arterial tension! Dr. Allbutt says it is especially in cases of interstitial nephritis that these attacks are most common. The kidneys I exhibit to-night are practically those of interstitial nephritis.

In my patient, owing to his age, the dyspnœa finally culminated in death, as he was an old man, and the heart was fatty and dilated. Nevertheless, I was enabled by treatment to prolong his life for several days. Towards the last, from cardiac failure and pulmonary congestion, râles indicating œdema were heard. In the course of interstitial Bright's disease transient dyspnœa is frequently noticed. Occasionally this can be understood from various pulmonary or cardiac lesions. In

the absence of these this uræmic asthma is a reasonable explanation, and it is to be diagnosed by the presence of the symptoms of asthma, and the absence of bronchial spasm. The paroxysm occurs because the blood cannot reach the air, not because the air cannot reach the blood. I have seen other cases in which treatment has averted the danger, and the physiological action of remedies lends us aid in confirming the truth of the theory. This is not the place to speak of such evidence. The determining causes are chiefly mental, mainly emotional in their character, as, for instance, care, sudden excitement, and the like, and, on the other hand, an attack may be precipitated by over-indulgence in food and the introduction of imperfectly assimilated albuminoid principles into the circulation, or, even without the intervention of dyspepsia, more food may be ingested than can be applied to the conservation of the tissues or eliminated as waste.

Dr. Tyson called attention to the openings of the ureters into the bladder, in comparison with those of a specimen lately presented by him. In the latter the ureters were closed, or almost so, while it was evident also that several calculi had descended into the bladder. In the specimen exhibited to-night the orifices of the ureters are large enough to admit the little finger. This, Dr. Tyson thought, could be explained by the condition of the two bladders; in Dr. Bruen's specimen there is a ribbed bladder, the result of an inflammatory process, in which there exists a tendency to back the urine into the ureters, causing their dilatation; in his case this backing of urine from the bladder into the ureters did not occur, as the point of obstruction was above. *March 11th*, 1880.

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4. *Supra-renal capsules, kidneys, and enlarged lumbar glands from a case of Addison's disease.*

Presented by Dr. Frederick P. Henry.

The clinical history of this almost typical case of Addison's disease is very meagre in details, owing to the fact that I saw the patient, a female, æt. about 50, but once before her death. The facts obtained are, however, of considerable interest. The woman was admitted to the Episcopal Hospital on the afternoon of May 26, and died on the morning of May 28. While examining her on May 27 I was struck with the peculiar sallowness of her complexion, and asked her whether

she had been as dark-colored when in health. She replied that her skin had darkened in color since her illness, which dated from August, 1879. The color could not by any stretch of imagination be compared to *bronze* of any kind, but rather resembled the hue of jaundice in slight amount occurring in a person of dark complexion. The patient was naturally a brunette. The conjunctivæ were natural in color, and although in describing the color of the skin I have spoken of jaundice, I entertained no suspicion of the presence of that affection. As is not infrequently the case in this remarkable disease, many cases of which are of traumatic origin, the patient was able to assign a definite period to the commencement of her illness. The attack began in August, 1879, with weakness and pain in the back immediately following the occupation of weeding her garden. These local symptoms continued with greater or less intensity ever since, but the pain was latterly most severely felt in the left lumbar region, in which situation there was also a great degree of tenderness on pressure. About three months before admission she had had a violent attack of enteralgia lasting for several days, the pain being greatest in the hypogastric and right and left inguinal regions. The woman was thin as to the arms and thorax, but not at all emaciated, and the abdomen was full and round, with considerable fat in its walls. The tongue was slightly coated. She suffered from occasional vomiting, and the bowels were, as a rule, constipated. The abdomen was more or less tender throughout, the tenderness increasing as the left lumbar region was approached. There was some cough, and physical examination of the thorax revealed slight dulness at right apex before and behind, prolonged expiration, and increased vocal resonance. The act of sitting up in bed while the thorax was examined was followed by considerable exhaustion. The pulse was soft and compressible, though full. The general intelligence was good. Sleep was poor. The pupils were natural.

Examination of the urine served rather to mislead. It contained blood-corpuscles and uric acid crystals; the latter visible to the naked eye in the form of red-pepper grains, and under the microscope in the form of rosettes. These characters of the urine, taken in connection with the fixed pain in the side and the severe paroxysm of abdominal pain three months before, led to the conclusion that the case was one of nephrolithiasis.

On visiting the hospital on May 28 I was surprised to hear of the patient's death, as there was nothing in her condition the day before

to indicate the near approach of a fatal termination. I may say in this connection that this sudden mode of death in Addison's disease is not infrequently met with, and is referred to by Wilks and Moxon,[1] as follows: "The process is chronic, and lasts often several years. The patients die generally slowly by asthenia, but some have gone off suddenly by rapid sinking and syncope." Greenhow refers to the manner of death in almost the same words, so that it is presumable that he was quoting from Wilks and Moxon, or *vice versa*. He says,[2] "Death takes place by asthenia, and sometimes, at the last, almost suddenly, apparently in syncope."

At the autopsy, quite a thick layer of fat was found in the connective tissue of the abdominal walls. The blood was fluid, and on removing the liver it poured out of the inferior vena cava in large amount. There was none of the dryness and translucency of tissue so constantly met with in severe cases of anæmia. In the mesentery of the small intestine, in the meso-colon, and in the gastro-hepatic omentum, a few miliary tubercles were found; also on the peritoneal surface of the first portion of the duodenum. The mesenteric glands were greatly, and the lumbar glands enormously, enlarged. Along the vertebræ these latter were firmly united by dense masses of connective tissue in which the solar plexus and semilunar ganglia must have been imbedded, for a careful and persevering attempt to dissect out these structures was unsuccessful. The degree of this overgrowth of connective tissue, in connection with the enlarged glands, may be estimated to some extent when it is mentioned that I found it impossible to remove the pancreas except in fragments. This being the case with regard to so prominent an organ, it is not to be wondered at that the semilunar ganglia and solar plexus could not be found. Both suprarenal capsules were greatly enlarged, and hardened. They were firmly adherent to the neighboring tissues through the medium of dense connective tissue, and were removed with much difficulty. There was no greater irregularity of their surfaces than could be attributed to ruptured adhesions. The centres of the capsules, particularly of the left, were paler in color and softer than the surrounding portions, which gradually shaded from a pale red about the slightly yellowish centre, to a deep red at the periphery. The naked-eye appearances were such as to indicate that the proper tissue of the organ had been

[1] Lectures on Path. Anat., Phila., 1875.

[2] Croonian Lectures on Addison's Disease.

replaced by a new growth of a fibroid structure. All distinction between cortical and medullary substance, and all trace of a central cavity, were obliterated. In order to show the comparative relations of size and situation, the capsules were not separated from the kidneys, and consequently there is no record of their weight. It is, therefore, difficult to convey an idea of their size. Both capsules retained their normal cocked-hat shape. The left, the larger, was closely adherent to the kidney, which was of normal size, and was estimated by those who saw it to be fully its equal in bulk. The right was looser in its attachment to the kidney and much smaller. It was calculated by one of the members of the Society, a member of the Committee on Morbid Growths, that the left capsule was twenty and the right five times larger than normal. The kidneys and spleen appeared normal. At the apex of the right lung was a slight degree of induration and some puckering of the surface. The induration was very superficial. Neither tubercle nor cavity was formed. Similar but still slighter changes were found in the left apex.

In the opening sentence of this paper I spoke of this case as being *almost* typical; and this expression needs some explanation. By a typical case is meant one in which the sole naked-eye lesion is in the supra-renal capsules. In this sense, the case which I reported in vol. v. of the Pathological Society's Transactions is a typical one. It must be borne in mind, however, that there is overwhelming evidence to prove that although, in a typical case, the disease begins in the capsules, yet many of the symptoms, perhaps among them the peculiar discoloration of the skin, do not arise until the neighboring plexuses and ganglia of the sympathetic are involved in the inflammation. The changes in these organs are often only to be perceived with the aid of the microscope. Although in this definition of a typical case I follow the high authority of Greenhow, who has collected one hundred and one cases in which there existed no other organic lesion than that of the capsules "which could be of the slightest importance," nevertheless I think it is extremely doubtful whether the coexistence of tuberculosis in any degree should forbid the classification of a case under the head of typical. According to Wilks and Moxon, of fifteen cases exhibited at the London Pathological Society, twelve showed more or less tuberculous disease of the lungs; in two the lungs were not examined; and only in one case are they said to have been found healthy. The enlargement of the lymphatic glands cannot be regarded as a complication unless as regards its unusual degree, for Greenhow states

that in "almost every case" he had seen there were some enlarged lymphatic glands in the "dense connective tissue surrounding the capsules." He also states that "the mesenteric and retroperitoneal glands in the neighborhood of the capsules are generally enlarged." Enlargement of the spleen has been reported in a number of genuine cases, but was not found by Greenhow in any of his own.

For proof that the melasma supra-renale originally described by Addison is dependent upon a uniform lesion, and for an exact description of that lesion, I refer the reader to Greenhow, both for the sake of brevity and in the hope that thereby some may be induced to read his admirable monograph upon Addison's disease.

As showing the rarity of this affection, I may refer to the fact that in the nine volumes of the Transactions of the Pathological Society of Philadelphia there is but one case recorded, and that was exhibited by me in 1874. *June 10th*, 1880.

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### 5. *Foreign body in the urethra and bladder.*

Exhibited by Dr. C. B. Nancrede.

The patient, a man of about 50 years of age, had been admitted to the Episcopal Hospital for a supposed medical ailment. On the second day after admission Dr. Nancrede was asked to see the case. He found him passing very little urine, and, upon examination, an ordinary penholder was found lying in the urethra, partly in the bladder and partly in the spongy urethra, the end being discernible to the touch in the perineum. The size was about No. 25 French scale. It was found to be impracticable to withdraw the foreign body by any variety of urethra forceps, and perineal section was resorted to. With the exception of some cystitis, the man did well; no fistula resulted. The section was made in the median line, partly through the bulbous portion of the urethra, Dr. Nancrede thought, yet no severe hemorrhage occurred. *September 9th*, 1880.

### 6. *Vesical calculus.*

Presented by Dr. C. B. Nancrede.

C. S., aged 5 years, had been suffering from difficult and painful micturition for about three years at the time when I was asked to see him. Two years previously he had been taken to a hospital in this city, where the hypertrophied condition of his prepuce, instead of exciting suspicion of a vesical calculus, only induced the surgeon on duty to circumcise him. In the latter part of January, 1881, he was admitted to St. Christopher's Hospital under the charge of my friend, Dr. Wm. H. Bennett, who immediately suspecting the nature of his ailment, sounded him and readily detected a stone.

He kindly requested me to operate for him, which I accordingly did on February 9, 1881. No difficulty was experienced in reaching the stone, but it was only after prolonged efforts, consequent upon repeated failures, that, after enlarging the wound in the bladder, I finally extracted this unusually large stone for so young a child. It measures one inch and a half, less a sixteenth, in the longest diameter, by one inch in width, by five-eighths of an inch in thickness, and weighed two hundred grains. *March 10th*, 1881.

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## *b.* FEMALE ORGANS OF GENERATION.

### 7. *Soft fibroma of uterus.*

Exhibited by Dr. H. F. Formad.

I obtained this specimen and the history of the case through the kindness of Dr. S. A. S. Jessop, of the Philadelphia Hospital, from a case in his private practice in Armstrong County, Pennsylvania.

Mrs. A. C., aged 30, has been confined nine times; only one child living; the rest were either still-born or died during delivery. Owing to a malformation of the pelvis, forceps were always used.

In the summer of 1880 she was last delivered of a still-born child. After the removal of the placenta the doctor made an examination, and discovered a tumor attached loosely to the mucous membrane of the uterus, which by gentle traction was easily removed. Hemorrhage

was profuse; the contraction of the uterus did not take place for hours, and then only after administration of enormous doses of ergot.

The patient subsequently recovered and regained perfect health.

The tumor was lobulated, had a distinct capsule, and measured about six inches in its largest diameter; in some parts it was deep red in color, and was of elastic, moderately hard consistence.

Microscopic examination showed the tumor to be made up exclusively of areolar connective tissue, with large areas infiltrated with blood. Portions of the growth not infiltrated with blood showed a beautiful mesh-work formed by stellate cells, densely packed in some places, in others forming a loose mesh-work with prominent nuclei at the nodal points. Bands of fibrillar tissue were scarce, forming in places a concentric arrangement around bloodvessels. These appearances lead to the diagnosis of a *true soft fibroma*.

*December* 23*d*, 1880.

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### 8. *Hypertrophy of nymphæ.*

Exhibited by Dr. B. F. Baer. (Permission having been on motion accorded him, Dr. Baer's paper will appear in the *Obstetrical Journal.*)

Dr. Formad said that he had made a microscopical examination of a section of the specimen presented by Dr. Baer. The surface was covered by the normal cuticle of a mucous membrane somewhat hypertrophied, owing to exposure to the air. The rest of the growth consisted of succulent fibrous tissue, so that he would call it a polypoid fibroma.

Dr. S. W. Gross asked whether Dr. Formad had noticed anything peculiar about the lymph-spaces.

Dr. Formad replied that in the single section examined by him nothing unusual was noted with regard to these cavities.

*March* 10*th*, 1881.

9. *Polypoid tumor of labia.*

Exhibited by Dr. H. F. Formad.

Dr. U. M. Beachly, of Meyersville, Penna., sent me this interesting tumor for examination, accompanied by the following history:—

Removed from a married lady, aged 38, robust health, weight 210 pounds; eight children, all living. The tumor has been developing for four years. Never gave her much pain. It was attached by a pedicle to the right labium pudendum. The pedicle was two and a half inches in length and one-quarter inch in diameter, having a peculiar appearance, resembling the trachea of a small animal. The tumor when removed was two ounces in weight, in external appearance resembling a cauliflower growth, having an uneven surface, covered by a skin, in consistency soft and elastic, and in color bright red. On one side of the tumor was a scab which bled freely when removed.

Upon microscopic examination, the tumor shows the structure of a lipomatous myxoma.

Dr. S. W. Gross said that such tumors were very rare, but that Virchow had delineated a pedunculated myxomatous tumor of the labium majus of much larger size than the one just presented by Dr. Formad, and that Paget refers to several examples.

*October 28th*, 1880.

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c. MALE ORGANS OF GENERATION.

10. *Hypertrophy of the prepuce.*

Presented by Dr. J. E. Garretson.

Patient a native of the island of Barbadoes; aged 60 years. Associated with the enlargement and induration of the prepuce there was an apparent relaxation of the tissues of the scrotum, that appendage being some ten inches in length and of very little less width. The hypertrophy of part shown commenced several years back. When in place over the glans the full circumference of the structure seemed involved; when retracted the thickening was seen to be confined to one side. Removal was not attended by any hemorrhage. The case occupied the ordinary time in healing of an operation for circumcision.

*October 28th*, 1880.

11. *Large spindle-celled sarcoma of the sheath of the penis.*

Presented by Dr. W. G. Porter.

John H. Henton, black, widower for the second time, New York, æt. 44, temperate, was admitted to the Presbyterian Hospital on the 25th of August, 1880, and gave the following history. His health had always been good. He had never had any venereal disease, and he was the father of three healthy children. In March last, while employed as a porter in a large retail clothing-house, he was engaged in moving a very heavy package from a truck to the cellar, and while sustaining an unusually heavy weight, he felt something give way in his groin. He immediately dropped his end of the package, vomited, lay down, and felt so sick that he had to stop work. He went home and remained in bed for several days. He then returned to work, but in a few weeks noticed a tumor in the right inguinal region, which rapidly increased in size. Supposing that he was ruptured, he applied to an instrument-maker for a truss, who gave him a suspensory bandage. The tumor from this time enlarged very rapidly, and he was soon compelled to stop work, and applied to me for relief. On examination, a large tumor was found filling up the right groin, surrounding the penis, and extending deeply into the perineum. Neither testicle was implicated by the growth. The tumor was heavy and exceedingly hard. There were no evidences of fluctuation about it, and throughout the tumor felt as hard as a fibroid. There was no difficulty in passing water, and a bougie or catheter readily entered the bladder, and the course of the urethra could be easily demonstrated on it. As the man was incapacitated for work and the tumor was increasing in size, its removal was advised, and on the 27th of September, with the advice and assistance of the surgical staff of the hospital and of Prof. S. D. Gross and Dr. S. W. Gross, the tumor was removed. An oblique incision was made over the long axis of the growth, and after a rather tedious dissection by knife and finger the attachment was found to be to the right side of the sheath of the corpora cavernosa of the penis; only at one point, and that a small one, was the right cavernous body itself involved. The hemorrhage was slight and was controlled by the ligation of several bleeding points. There was some capillary oozing, which was controlled by the application of hot water. The wound was carbolized, a drainage-tube inserted, the

lips of the wound approximated by stitches, a compress and bandage applied, and the patient removed to bed. He soon came out from the ether, showed no evidences of shock, and seemed to be in good condition. In the early morning of the next day his belly became tympanitic, and he complained of the wound being tender and sore. The compress and bandage were removed, as there had been no oozing, and the wound was lightly dressed. He passed his urine without the assistance of the catheter. A turpentine enema brought away large quantities of gas. The following day the penis was swollen and the prepuce very œdematous; the belly tympanitic again. Enemas and the passage of a rectal tube failed to give relief. The patient suffered considerable pain; the tongue was dry; the edges of the wound dry, glazed, and erysipelatous; no discharge from the wound or drainage-tube; the penis became gangrenous; the belly remained tympanitic, and the patient died, exhausted, on the 1st of October, four days after the operation. Autopsy showed no evidences of peritonitis or cellulitis, though there was some very fetid gas in the peritoneum, but nothing but the local lesions already detailed to account for death.

Dr. Seiler said that he had made a number of sections from the tumor which had been sent to him by Dr. Porter, and on examination had found them to consist of spindle- and round-cells of the large variety, the former predominating, so that the growth should, in his opinion, be described as a large spindle-celled sarcoma.

Dr. S. W. Gross said that through the kindness of Dr. Porter he had seen the tumor before its removal, and had then regarded it as a sarcoma. The operation plainly showed that it arose from the sheath of the corpora cavernosa, which is continuous with the superficial perineal fascia, as was first demonstrated by Dr. Buck, of New York, and that it had invaded the tunica albuginea of the corresponding body the extent of about two-thirds of an inch. He had looked up the literature of the subject, and the only reference he could find to a similar growth was a case recorded by Kochler in the *Charité Annalen*, 1878, and quoted in the *Index Medicus* for July, 1880. In a late posthumous work of Demarquay, published by two of his pupils, and entitled "Maladies Chirurgicale du Penis," no mention is made of any such growths. From this he would infer that sarcoma was a very rare affection in this region, and that in all probability this case of Dr. Porter's was the only one that had ever been reported in this country.

*Oct. 28th*, 1880.

### 12. *Sarcoma of testicle.*

Exhibited by Dr. H. F. Formad.

This tumor was removed from a young man by Professor Agnew in the surgical clinic of the University Hospital, in October last. I failed to obtain a complete history of the case, and learned only the following from the gentlemen of Dr. Agnew's clinical service. The patient was about 17 years of age, and in apparent good health. The tumor developed within one year, slowly at first, but later growing more rapidly, reaching, at the time of the operation, the size of a large fist. At no time was there any pain; and the only inconvenience experienced by the patient was from the weight and the size of the tumor. The skin was not involved.

On examination, after removal, the tumor showed itself to be developed from the tissue of the left tunica vaginalis testis; the structure of the testicle was not at all involved, but the whole testicle of that side was simply pushed up into the upper part of the sac, and was much atrophied. The tumor had a thin capsule, was elastically hard in consistence, and on section presented a white fibrillated appearance. Microscopical examination revealed the typical structure of large spindle-celled sarcoma.

Dr. S. W. Gross thought that in this particular instance the tumor had started in the connective tissue between the dartos and tunica vaginalis testis. Sarcoma may arise from the connective tissue of any part, but he considered that the site of the present one was unusual. He also related a case seen by Prof. Gross, presenting the following history. The gentleman had been operated upon in Kentucky for a sarcomatous growth, as was then thought, connected with the testicle, which organ, he stated, had been removed at the operation. Upon careful examination, Prof. Gross determined that the recurrent growth for which he had been consulted was in reality a diseased testis. The former operator had doubtless removed a growth like the present one, which resembled in form a tumor of the testis, and which probably sprang from the connective tissue. Prof. Gross, in operating for the recurrent growth, tied the cord, thus demonstrating the presence of the gland. The wound healed slowly, recurrence took place, despite the use of the actual cautery, etc., and death finally ensued.

Dr. H. F. Formad had had no opportunities of observing patients

affected with sarcoma of the testis, but it could not be a very rare disease, since he had had sent to him for examination in the past few years no less than nine specimens. Of these, two sprang from the connective tissue; four were of the round-celled variety, and were associated with more or less cartilage; two consisted of ordinary spindle-cells; while one was of the large spindle-celled variety. One of the specimens seemed to have had its origin in an inflammation set up by the injection of iodine into a tumor of the testis. Dr. C. T. Hunter had been consulted some few months subsequent to this, when he found that a number of large nodular growths had formed, which induced him to castrate the man. Microscopically these masses were seen to be sarcomatous, while the bulk of the tumor consisted merely of new inflammatory tissue. In this case, thirteen months after the operation, the glands of the neck became involved. In another case, where he had been requested to examine, post mortem, the supposed tuberculous testis of a man dead from phthisis, he had, to his surprise, found that the organ was affected with sarcoma.

Dr. S. W. Gross asked Dr. Formad whether in his examinations of the round-celled variety he had ever found any traces of gland-structure.

Dr. Formad replied that he had in one case.

Dr. J. H. Brinton recalled the case of a prominent gentleman of this city upon whom he had operated some three or four years back for sarcoma of the testis. The wound healed readily, but in nine or ten weeks the retroperitoneal glands became involved, a little later those of the neck, and death rapidly occurred. *January 13th*, 1881.

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13. *Sarcoma of testicle.*

Exhibited by Dr. J. H. Brinton.

J. L., æt. 40 years, a farmer by occupation, came under my care in May, 1880. He asserted that the enlargement of his testicle had commenced three years previously. No history of traumatism or other exciting cause could be ascertained. Castration was performed May 4, 1880, and the patient died of pyæmia, June 1, 1880.

A microscopic examination by Dr. S. W. Gross showed that the tumor consisted of large spindle-cells, with here and there a fibrillated inter-cellular substance. The seminal tubules were dilated, deformed, and lined with columnar epithelium. *January 13th*, 1881.

14. *Two Cases of Round-Celled Sarcoma of the Testicle, with Local Recurrence and Secondary Deposits after Castration.*

Presented by Dr. S. W. Gross with remarks.

Case I.—A laborer, 35 years of age, was sent to me on the 8th of June, 1880, by Dr. T. W. Taylor, of Kennett Square, on account of a smooth and uniform enlargement of the right testis, which was of the size of a large cocoa-nut, of an elastic and here and there pseudo-fluctuating consistence, and the seat of occasional pain. About three years and a half previously the testis became greatly swollen from the man being jammed between the wheels of a wagon; but the inflammation gradually subsided, leaving, however, the organ about double the size of its fellow. Fifteen months ago it began to increase, until it finally attained the volume above mentioned. After castration the albuginea was seen to be pervaded by immense tortuous veins; but that tunic was not thickened, nor was the cord involved. Section was attended with the escape of a dirty yellowish fluid, and the cut surfaces were of a translucent grayish tint, and mottled by large areas of blood-stained broken-down tissue and by small spots of caseous degeneration, and they also contained a few cysts. The entire upper portion of the tumor was converted into a cheesy mass as large as a billiard-ball. Microscopical examination disclosed a small round-celled tissue, the intercellular substance of which was granular. There was no trace of seminal tubules.

At the expiration of three months the man began to complain of pain in the belly, and soon afterwards Dr. Taylor discovered a tumor as large as an orange just below and to the right of the umbilicus. The suffering soon became severe and constant; temporary paralysis and œdema of the lower extremities ensued; the bowels could only be moved by purgatives; and nausea and vomiting, which came on every afternoon, continued throughout the night unless he was fully under the influence of morphia. When I saw him, on December 4, the symptoms had not abated. He was growing thin, had no appetite, and his face was becoming icterode. A small recurrent growth occupied the scrotum. The abdominal tumor, which I was informed had doubled in size in the last ten days, was as large as a child's head, filling the right lumbar and nearly the entire umbilical region, and had an elastic feel. Exploration of the chest disclosed the physical signs of second-

ary deposits in the upper lobe of the right lung. Death ensued on the 20th of December; but an examination of the body was refused.

Case II.—A farmer, 33 years of age, consulted Prof. W. H. Pancoast on account of a tumor of the right testis, which he said was of four years' duration, and had developed in consequence of a blow received in climbing a fence. Its consistence was elastic, its outline smooth and pyriform, and its volume equalled that of a small cocoa-nut.

The organ was removed in the usual way on the 24th of February, 1880. The cut surfaces had a medullary aspect, and the minute structure was that of a granulation sarcoma, with entire disappearance of the seminal tubules.

The wound closed promptly, but in two months there was recurrence in the stump of the cord. Early in September the growth had attained the size of a fist, when Dr. Pancoast removed it by laying open the inguinal canal and ligating the cord at the internal ring. The wound healed in four weeks, but on his return to the hospital of the Jefferson Medical College, October 22, it was found to have opened, and there was a fungous mass in the groin as large as an orange, which was scraped away, and the parts freely touched with chloride of zinc. Shortly afterwards a tolerably firm and painful tumor was detected in the retroperitoneal glands of the corresponding side, on which account nothing further was done in the way of surgical interference. At the date of his discharge, December 1, the abdominal tumor was as large as a child's head, and the glands of the left supraclavicular fossa were as big as an egg. The man was decidedly anæmic, and he had been suffering for several weeks from gastralgia, loss of appetite, and almost constant nausea and vomiting.

*Remarks.*—The cases which have been presented to the Society this evening possess many characters in common, and two, namely, that of Dr. Pancoast and my own, are especially interesting as being examples of reproduction of the disease in the abdominal lymphatic glands and of local recurrence shortly after operation.

Implication of the glands is a remarkable feature in connection with sarcoma of the testis when considered in relation to the immunity of the glands in sarcoma of other organs. In sarcoma of the breast, for example, as I have pointed out in my "Treatise on Tumors of the Mammary Gland," involvement of the axillary glands is not met with during life, nor does post-mortem inspection disclose that the deep glands are converted into metastatic tumors. In the disease under consideration, on the other hand, secondary glandular growths are more com-

mon than they are in carcinoma of the testis, so that they afford no aid in the differential diagnosis, as is the case in carcinoma and sarcoma of other organs. From an examination of 40 examples of sarcoma of the testis which I have collected from various sources, I find that the histories are complete in 26. Of these, three were well after castration for an average period of six years, two being free from recurrence for two years, and one for fourteen years. Three pursued a natural course, and post-mortem inspection showed secondary tumors in the lumbar glands, bones, and subcutaneous tissue in one, and visceral tumors, without glandular enlargement, in two. Twenty were castrated; of these, one—the case of Pancoast—is still living, with local recurrence and enlargement of the lumbar and supraclavicular glands, and 19 are dead, with glandular implication and visceral deposits, especially in the lungs and osseous system, in 16. Hence, of the 26 cases, in 18, or 69 per cent., the retroperitoneal glands were the seat of secondary deposits; and I find that they are involved in 62 per cent. of the examples of carcinoma. In several of the cases the absence of glandular affection may be explained, as is pointed out by Mr. Butlin in a valuable paper on the subject in the *Lancet*, August 28, 1880, by the short duration of the disease.

The great frequency of the development of secondary growths in the retroperitoneal glands into which the lymphatics of the testis empty, tends to show that sarcoma originates from the endothelial cells of the large lymph spaces which surround the seminal tubules, rather than from the membrana propria of the tubules.

Local reproduction in the stump of the cord or in the adjacent tissues, which constitutes the second interesting feature of the disease, is so uncommon that it was met with in only five of the twenty-three cases in which the histories are complete after operation.

As specimens of sarcoma of the testis have been so rarely exhibited to the Society, I take this occasion to analyze still further the cases which I have collated and to compare them with those of carcinoma. Seventy-five per cent. of all examples of sarcoma occur before the forty-first year, and 25 per cent. before the eleventh year, three having been observed respectively at eight, ten, and eleven months. Fifty-six per cent. of all ages of carcinoma are met with before the age of 41, and 6 per cent. before the age of 11. In sarcoma the epididymis is more early implicated, its growth is more rapid, and its volume is greater; the scrotum is not adherent, and it ulcerates in only 3 per cent. of all instances; and the cord is involved in the dis-

ease in 35 per cent. and ulcerated in 9 per cent. In carcinoma the scrotum is adherent in 36 per cent. and ulcerated in 9 per cent. of all examples, and the cord is implicated in 63 per cent. of all specimens. In sarcoma the tunica albuginea is rarely thickened, while that occurrence is the rule in carcinoma. In the former affection both testes are not uncommonly affected, while one alone is involved in the latter disease. Finally, the presence of cartilage is so frequent in sarcoma, and so rare in carcinoma, that it goes far to establish the diagnosis.

Of the 40 cases, 22 were of the round-celled, 15 of the spindle-celled, and 3 of the mixed variety.

The prognosis of sarcoma is eminently unfavorable. Thus, of the three cases which pursued a natural course the average duration of life was less than ten months. Of twenty-three patients subjected to operation, three were living at the date of the last reports for an average period of six years (and, strange to say, they belonged to the round-celled variety), one was still alive with local and general reproduction, and nineteen died of generalization of the disease, their average life having been twenty-seven months. Hence castration not only prolongs life, but may effect a cure. The total duration of life from the first observation of the disease until its termination after operation averaged eighteen months for the round-celled and twenty-eight months for the spindle-celled variety; and the former was followed by metastatic tumors in eighteen per cent. more of instances than was the latter.

*January 13th*, 1881.

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### *d.* MAMMARY GLAND.

### 15. *Ulcerated scirrhous carcinoma of the mammary gland occurring at an early age.*

Presented by DR. SAMUEL W. GROSS.

The specimen before you is the entire left breast, which I amputated this morning. The patient, a married, prolific female, 27 years of age, first noticed, twenty months ago, a nodule to the outer side of the nipple, which gradually increased until the entire gland was involved. The glands of the axilla were observed to be enlarged in eight months, the nipple began to retract in twelve months, and the skin was superficially ulcerated in seventeen months, from the commencement of the

disease. During the last four weeks the axillary glands increased to three times their former size, became densely indurated, and fixed to the axillary border of the great pectoral muscle and the side of the chest; the superficial ulceration was converted into a deep, crater-like sore; several nodules appeared in the skin at the inner circumference of the growth; and the mass attached itself to the pectoral muscle. There was no history of heredity, trauma, or psoriasis or eczema of the nipple.

The operation was made as thorough as possible by removing the entire gland along with its skin and subcutaneous fat, the greater portion of the pectoralis major muscle, and the involved lymphatic glands.

The interest of the case consists mainly in the comparatively early age at which the disease was first observed—namely, twenty-five years and four months. In my "Practical Treatise on Tumors of the Mammary Glands," which has just appeared from the press, I state that out of one hundred cases of carcinoma of the breast of which I had a record the earliest age that I had observed was twenty-eight years. Since the chapter on carcinoma was written I have met with twenty-six additional instances, and the one now recorded was the only one noted before the age of thirty. *September 9th*, 1880.

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### 16. *Adenoma of the breast.*

Presented by Dr. Morris Longstreth.

The specimen was obtained from a patient in the Pennsylvania Hospital, named Ann Williams, colored, æt. 53, widow, washerwoman, who was admitted October 7, 1879, under the care of Dr. R. J. Levis. Her family and personal history was good. She had been treated in the hospital previously for rheumatism. Eighteen years ago, while washing, she ran a needle into the right breast at the nipple; it caused but little pain, and she had forgotten it until, two months after, she noticed a slight discharge, which was bloody, from the nipple. Two or three months later a hard lump was found in the breast, on examination. The discharge ceased after a time, but the breast steadily and very slowly increased in size. Eight years ago (ten years subsequent to the injury) a sore broke out below the nipple, which has never healed; a year ago another sore formed to the outer side of the areola,

and there has been a continual slight discharge from both openings. There have been darting and burning pains in the breast for years, and for the last few years the pain has been constant. The patient was married twenty-five years ago; has had three children (one of whom died of croup, the other two were still-born), all prior to the breast trouble.

On admission she was seen to be stout and healthy-looking, and felt well, with the exception of the pain, which prevented sound sleep. She wished for an operation on account of the pain in the breast. There was found a tumor in the right breast, which was very large, prominent, and irregularly conical in shape. There were seen the two openings which the patient had described—the one below, the other a short distance to the outer side of, the nipple. The breast was removed (Dr. Levis operated) by two incisions, making an elliptical figure over the tumor from right to left and slightly downwards, about six inches in length and two inches at the widest part. The mass was about the size of two fists. The axillary glands were not enlarged.

*Description of the tumor after its removal.*—The mass presented a very prominent, somewhat conical shape, covered by an elliptic-shaped flap of black skin, in the middle portion of which were the nipple and the two ulcerations. The nipple was very slightly projecting, having a soft, delicate feeling to the touch. The ulcerated openings were of small size, scarcely admitting the tip of the little finger; they were seated at the bottom of slight depressions in the surface of the breast, and were surrounded by areas of whitish, cicatricial tissue, contrasting markedly, both in color and consistence, with the adjacent normal black-pigmented skin; their edges were smooth and tended to inversion, and showed no appearance of fungous granulations. A probe passed into these openings entered a narrow sinus leading a short distance into the substance of the tumor; a slight amount of pressure brought up a little thick matter, of a pinkish color, similar in character to that discharged previous to the operation.

A large portion (probably a fourth) of the mass removed was composed of normal adipose tissue, situated largely in the direction of the axilla. The tumor itself was a rounded mass, somewhat flattened; a considerable amount of fat was laid over, and pretty firmly adherent to the firm, tense, shining, fibrous capsule enveloping the new growth. The tumor was lobulated, with the lobules well compacted. An incision, made from the posterior surface and carried to the region of the nipple, showed the large masses to be composed of a homogeneous,

pinkish-white tissue, subdivided into smaller nodules by bands of connective tissue of a varying width. The surface of the section was finely granular, and there were seen numerous small cysts seated in the interior of the nodules, more numerous towards the nipple portion of the growth; these cysts contained a thin, blood-stained fluid, varying, however, in its depth of color. At the lower border of the tumor, in the neighborhood of the opening, on the skin below the nipple, were several large-pea-sized cysts filled with blood, and having thick, fibrous walls.

The tumor, after the fat was removed, measured five inches in length, two inches in breadth, and three inches in thickness.

*Dec.* 11*th*, 1879.

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17. *Two cases of true adenoma of the mamma, with brief remarks upon the histology and general pathology of that affection.*

Presented by Dr. Samuel W. Gross.

The specimen which has just been exhibited by Dr. Longstreth is very similar in its main features to one which he presented to the Society last spring. In both the tumor developed after the thirtieth year; in both it ulcerated and discharged a sanious fluid; and both had existed for eighteen years before their removal. In addition to other points of likeness in their gross appearances, they were pervaded by cysts, and were found, under the microscope, to be made up of cystic acini and ducts lined with columnar epithelium. From the predominance of ducts, they are both to be classed as tubular adenoma, which represents a common variety of this type of new growth.

Since the appearance of a paper from my pen on "True Adenoma of the Mamma," in the *American Journal of the Medical Sciences* for October, 1879, I have had an opportunity of examining microscopical sections of two additional examples. As adenoma is, next to myxoma, the most uncommon of all the neoplasms of the breast, I avail myself of this opportunity to show you the slides, with their histories, and present you with a summary of what I have deduced from the study of seventeen cases of this little-understood affection.

Case I.—A mulatto girl, æt. 16 years and 7 months, single, and the subject of regular but painful menstruation, noticed a tumor in the upper portion of the mamma, which caused her no inconvenience, but

led her to seek its enucleation, because "she did not like to have a lump in her breast." On its removal, three months subsequent to its appearance, by Dr. Armistead Peter, of Georgetown, D. C., it was found to be oval, nodulated, and to measure one inch and three-quarters in length by one inch and a quarter in breadth. At the expiration of twenty-eight months there was no recurrence.

Examination of a mounted section of the growth, which is numbered 6755, Surgical Section of the Army Medical Museum, shows, in addition to a few ducts, with appended acini, which are characteristic of the normal physiological condition of the breast at the age at which the tumor appeared, an immense number of closely-aggregated longitudinal and transverse sections of ducts, lined with spheroidal and cuboid epithelium, and provided with a central lumen. They are contained in a delicate, young connective tissue, and are, doubtless, offshoots from dilated acini.

Case II.—A married lady, barren, but menstruating regularly, discovered, at the age of 43 years, a small nodule in the upper and inner portion of the breast. It continued to grow, with periods of dull and sometimes lancinating pain, for three years, when her attendant punctured a prominent elastic nodule, which bled freely, and the slight wound took on ulcerative action. At the date of its removal, six years after its appearance, by Dr. John Sloan, of New Albany, Indiana, to whom I am indebted for the history and the microscopical section, the larger portion of the mamma was converted into an irregular, lobulated and firm tumor, the upper surface of which was converted into an excavated ulcer, which discharged a sanious fluid, and measured two inches in diameter. The nipple and axillary glands were normal. At the end of two years there was no tendency to recurrence.

The section discloses that the growth was composed, first, of enormously-dilated acini, lined by hypertrophied, cylindrical epithelium, a few of which contained vegetations composed of delicate connective tissue clad with epithelium of the same character, and, secondly, of ducts, many of which were beaded and cystic, and which were likewise lined by columnar epithelium. The latter preponderated to such an extent that the growth may be denominated a cystic tubular adenoma. The interglandular connective tissue was very delicate and sparse, and, here and there, the seat of an extensive small-celled infiltrate.

The term adenoma is applied by certain pathologists, as Cornil and Ranvier, Waldeyer, Luecke, Foerster, and Wilks and Moxen, to neo-

plasms in which there seems to be a simple exaggeration of the normal physiological structure of the breast, as the acini, along with their epithelium, preserve their normal form and size and arrangement, and are provided with a central lumen, the only difference being that the connective tissue is spare in the morbid formation. Of the five specimens that I have examined, the only one which approaches this description is the one removed by Dr. Peter, but the likeness to the normal structure is far from striking, the efforts of nature having been limited to the excessive production of glandular apparatus, without attempting to unite the acini into distinct lobules attached to excretory ducts.

The remaining sixteen examples that I have collated, and five were brought to the notice of this Society by Dr. Longstreth, Dr. Nancrede, and myself, all showed either marked enlargement of the acini or offshoots from the dilated and cystic acini, in the form of tubes or ducts, with aberrations in the characters of their investing epithelium. Thus, in ten instances the tumor was composed of excessively-dilated acini packed with polymorphous cells, which could be expressed from the cut surfaces of the growths as atheromatous moulds or plugs. It is to this form of adenoma that Labbé and Coyne, Malassez and Deffaux, and Duplay have incorrectly applied the term epithelioma. In the other six specimens the ducts predominated, and these arose, as I have demonstrated in the paper referred to, with the aid of a beautiful drawing by Dr. Shakespeare, from the dilated acini by a process of budding and extension into the scant connective tissue of the gland. In these cystic tubular adenomas the epithelium, instead of being polyhedral, assumes a cylindrical or columnar form, as is so well shown in the specimens of Dr. Levis and Dr. Sloan presented this evening. Hence it appears that true adenoma consists not only in a disorderly hyperplasia of the lacteal glands, but also in deviations in the form, size, and arrangement of the epithelium, on which account it is atypical in the strict sense of that term. The exception to this rule may be found in such specimens as that of Dr. Peter, which constitute the only one of the seventeen cases that I have analyzed in which there was any approach to the normal physiological standard.

Adenomas always occur singly: they develop as early as the sixteenth and as late as the fifty-ninth year, or at the thirty-fifth year on an average. The greatest number of cases are met with in prolific married women who menstruate regularly during the functional decline of the mamma. They usually arise without assignable cause, and the

general condition of the patients is excellent. They increase slowly, as a rule, and always by the apposition of new nodules to the original tubers. In exceptional instances they attain the volume of a double fist or fœtal head. In only 6.66 per cent. of all cases are the subcutaneous veins enlarged; the skin is discolored in 26.66 per cent. and adherent in 40 per cent.; the nipple is sunken, not retracted, in 13.33 per cent., and discharges a bloody or lactescent fluid previous to the detection of the tumor in 26.66 per cent.; ulceration occurs late in the disease in 40 per cent., and in nearly one-half of these there is fungous protrusion, without adhesion to the margins of the opening; and the lymphatic glands are the seat of irritative enlargement in 13.33 per cent. There is no pain whatever in two-thirds of the cases.

From these facts it follows that adenoma is characterized by its firm consistence, nodular outline, slow and equable growth; its occurrence in child-bearing females towards the thirty-fifth year; the limited discoloration and adhesion of the skin and ulceration late in the disease, and freedom from retraction of the nipple, prominence of the subcutaneous veins, attachment to the chest, and involvement of the axillary lymphatic glands. If the tumor has been preceded by a discharge by the nipple, the probabilities in favor of its being an adenoma are greatly increased.

With regard to the prognosis after operation, I will merely add that three patients remained well respectively for six, twenty-four, and twenty-eight months. One was alive at the end of two years, but with suspicious indurations near the cicatrice; one was well thirty-one months after the removal of a recurrent growth; while in one there was local recurrence and enlargement of the axillary glands in less than one year, and death ensued two years later, but there was no post-mortem inspection of the body. In the remaining eleven cases the history ceases with the recovery or death of the patient. Hence adenoma evinces a decided tendency to recur, but there is no evidence going to show that it infects distant organs.

*December* 11*th*, 1879.

18. *Extensive carcinosis of the osseous system and liver, consecutive to scirrhus of the mamma.*

Presented by Dr. Samuel W. Gross.

Rebecca H., 65 years of age, was admitted into the Philadelphia Hospital, on the 5th of July, 1879, on account of a supposed intracapsular fracture of the left femur. She was also suffering from a hard, painful tumor of the left mamma, which she attributed to a blow received a year previously, and there was also a hard, ovoid prominence at the site of the left frontal boss.

My colleague, Dr. Hatfield, removed the breast, along with a few hard and enlarged axillary glands, and the wound speedily healed. The skin of the entire body gradually became yellow; she lost flesh and appetite; the disease recurred in the axilla two months after the operation, and, ten days before death, she fell against her bed, and sustained a fracture of the left humerus.

On post-mortem inspection, February 11, the total life from the first observation of the mammary growth having been nineteen months, the cicatrix was sound and mobile, and the pectoral muscle was free from secondary nodules. The glands of the axilla were converted into a densely hard, knobby tumor as large as a lemon. All of the viscera except the liver were normal. The right lobe of that viscus, and especially its upper convex portion, was the seat of tubers, which varied in size from a pea to a walnut, and the largest of which were deeply umbilicated. In the lobe there were only two nodules.

The greater trochanter, neck, and head of the femur were infiltrated by carcinoma, and in the last two situations there were densely hard, round tumors, which were upwards of an inch in diameter. The neck of the bone was fractured at its point of union with the trochanter. The iliac glands were enlarged.

The left humerus was broken at the junction of its upper with its middle third. Its medulla had undergone carcinomatous degeneration, and projected a fungus into the surrounding soft tissues.

The frontal, parietal, and occipital bones were entirely infiltrated by the disease, and so soft that they could be readily crushed or broken. At the seat of the left frontal boss there was a tumor of central origin, which was of a biconvex shape, two inches in width, and half an inch thick at its centre. The left side of the occipital bone was occupied

by a similar but smaller growth. The dura mater was beset by nodules, which varied in size from a millet-seed to an inch transversely. The brain was healthy.

Minute examination of sections of the various growths disclose that they were tubular scirrhus. *February* 12*th*, 1880.

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19. *Hæmatoid myxoma of the breast.*

Presented by Dr. J. M. Barton.

Miss N. M., æt. 59 years, discovered in the early part of 1875, a small tumor in the right mammary gland. It grew but slowly, without pain. When removed in August, 1876, it had reached the size of a walnut, was distinctly encapsulated, and had developed in the cellular tissue rather than in the gland.

It fluctuated quite distinctly before removal, and was found to be filled with fluid blood, black and tarry. The wall from which the blood came was quite thin, and might readily have been taken for that of a simple cyst.

The tumor which I here present was removed at the German Hospital, March 2, 1880, is a little more than twice the size of its predecessor, and had been noticed by the patient since January, 1879. It has given but little inconvenience, being only occasionally the seat of sharp lancinating pains, intervals of three or four weeks having occurred in which the patient was entirely free from pain. The tumor was quite slow in its growth until six weeks ago, since which time it has doubled in size.

Previous to its removal it distinctly fluctuated, was immediately beneath the skin, to which it was attached, but encroached decidedly upon the mammary gland, was irregular in its outline and purplish in color, and had developed at the situation of the previous growth.

At the present operation I removed the entire gland with the tumor, as well as the attached skin.

On opening the growth it was found to resemble the previously removed tumor. *March* 11*th*, 1880.

*Report of the Committee on Morbid Growths:—*

"A section of the new formation is seen to consist of a delicate reticulum, made up of fibrils, spindle- and stellate-shaped cells; the

meshes formed by this reticulated structure are filled with a structureless, hyaline, intercellular substance, and an occasional lymphoid cell. Numerous bloodvessels are seen distended with blood. The histological structure indicates a myxoma. *April 8th*, 1880."

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### 20. *Carcinoma of the breast.*

Presented by Dr. John H. Packard. With report of microscopic examination of the same, by Dr. C. Seiler.

Susan C., æt. 50, unmarried, Irish, a large, well-developed woman, rather old-looking for her years, living comfortably as an upper servant in a wealthy family, noticed a tumor in her right breast some time last summer (1879). She does not recollect having ever sustained any injury to the part.

Becoming uneasy about it shortly before Christmas, she consulted her employer's physician, Dr. J. F. Meigs, who gave her a protective plaster and directed a tonic.

On the 12th of January she came to me by Dr. M.'s advice. The tumor had produced no visible alteration in the size or shape of the breast, but could be easily felt as a condensation and hardening of the part. It was not painful, or tender on pressure, but there was some "sticking" sensation in it after it had been handled. No alteration was discoverable either in the nipple or in the skin. Her general health was good in every respect. Menstruation regular.

I advised the continuance of the tonic, and the local application of a thin sheet of raw cotton, covered with oiled silk.

On the 29th of January the measurement of the diseased gland was eighteen inches, the tape being carried as accurately as possible around the largest vertical circumference. The sound one measured sixteen.

February 13th, the tumor measured seventeen and a half inches, and February 27th only seventeen.

On the 26th she had noticed a slight discharge of bloody liquid from the nipple. This still continuing, on the 27th I examined it under the microscope, and found the following elements: blood-corpuscles in large numbers; some bodies resembling the colostrum-corpuscles of milk; fine oil-drops; a few masses looking like epithelial casts of the lactiferous tubes; some cells of irregular shape, with nuclei

and nucleoli; a good many free nuclei were also noticed. Many of the nuclei contained two nucleoli.

Although the tumor had lessened in size, it had become decidedly harder and denser. There was no discoverable involvement of axillary or other lymphatic glands.

On the 19th of March I excised the tumor, with the entire breast, ether having been administered by Dr. A. V. Meigs, Drs. J. F. Meigs and Jesse Williamson being present and assisting. The incisions were made obliquely, so as to bevel the edges of the skin, and the wound was accurately closed with three hare-lip sutures and adhesive plaster. Carbolized cerate was used as a dressing.

She had no pain afterwards, except some in the arm from the constrained position in which it was kept. On the third day, the pins being removed, the wound seemed to have united firmly throughout. On the fourth day she got up to make her bed, and suffered only slight giddiness.

I would call attention to the effect of the oblique incisions, as enabling me to close the wound very accurately, excluding the air, and giving two broad surfaces for agglutination, thus promoting rapid union, and preventing the formation of pus even in the large cavity left by the removal of such a mass of tissue.

Dr. Seiler was kind enough to undertake the microscopic examination of the tumor, and to report his results:—

"The tumor of the breast exhibits under the microscope large distended acini, separated from each other by narrow bands of connective tissue. The epithelial cells lining the acini are proliferating, so as to fill in many instances the entire lumen, while in others the cell-plugs are seen to have undergone cheesy degeneration. Frequently the cells are seen to have broken through the limiting membrane of the acini, and to advance in pegs into the connective tissue. Numerous depots of small-celled infiltration are observed in the connective tissue. From these appearances I would pronounce the tumor to be a carcinoma having undergone cystic caseous degeneration." *March 25th*, 1880.

21. *Carcinoma of mammary gland.*

Presented by Dr. C. Seiler for Dr. Collins.

E. T. B., æt. 51. Tumor of right mamma. Growth had been noticed about one year; within a month had grown painful; removed. *May 27th*, 1880.

*Report of the Committee on Morbid Growths:—*

"A section of the tumor removed with the mammary gland shows it to be a scirrhous carcinoma; the histological structure consists of epithelial cells placed in alveolar spaces; the latter have their walls formed by fibrous tissue. *June 24th*, 1880."

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21. *Inter-acinous fibroma of mammary region.*

Presented by Dr. J. E. Garretson.

The tumor shown was removed from the superficial fascia overlying the right mammary gland of a lady aged 40 years. It is hard, lobulated, contracted. The concentration of the growth afforded indication for its removal. The time of development has been about two years. In the absence of anything self-explanatory the tumor is to be classified with the neoplasms.

Dr. S. W. Gross, in examining the specimen, thought that he could detect a distinct capsule, which would, of course, exclude the idea of carcinoma. As it had been kept some time, however, he might be in error, and would suggest the reference of the specimen to the Committee on Morbid Growths.

Dr. Garretson asked the President if he then considered it to be non-carcinomatous.

Dr. Gross replied, that if this appearance of a capsule was not produced by the condensing effects of the preservative agent, and if he understood Dr. Garretson rightly as to its not involving the structure of the mammary gland, he should most decidedly consider it to be non-carcinomatous.

*Report of the Committee on Morbid Growths:—*

"The tumor removed from the mammary region by Dr. Garretson, and referred to your Committee on Morbid Growths, is found upon

microscopic examination to consist mostly of dense fibrous tissue, in which are seen the remains of glandular acini and ducts in a state of atrophy. The growth is an inter-acinous fibroma.

*October 28th*, 1880."

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23. *Cystic tumor of the breast.*

Presented by Dr. Longstreth for Dr. Packard.

Miss ——, æt. about 45, a well-nourished person, and always in enjoyment of good health, with the exception of some functional nervous disorders, perceived during the summer of 1880 a small lump at the upper and inner border of the left mammary gland.

She suffered no pain except after it was handled, but was alarmed by its steady increase in size. It was removed on January 25, 1881; it had reached the size of an English walnut, and proved to be a cyst. During the operation, notwithstanding careful manipulation, the wall gave way at one point, and a quantity of liquid like thin milk was discharged; unfortunately, none of the fluid could be collected for examination. The mammary gland seemed to be healthy, and was left entire.

A microscopic examination was made, the section being taken from the border of the cyst, including a portion of its wall and the adjacent glandular tissue. The cyst-wall was seen to be made up of condensed fibrous tissue, in which the remains of glandular structure were apparent. The near-lying acini or tubules were found slightly dilated and their epithelial lining stained more deeply, and the cells appeared en larged or hyperplastic, as though from irritation of the adjacent cyst. The other portions of the gland seen in the section showed nothing abnormal.

*Remarks.*—It is unfortunate that none of the fluid could be collected for examination, but the exigencies of the operation prevented it, and at a later examination of the cyst no fluid was to be found in it. Attention was given to the intima of the cyst wall, but nothing was discoverable in its structure that gave any evidence of the nature of the cavity or its contained fluid. It seemed probable, from the appearance of the fluid and the condition of the neighboring acini, that the

cyst was to be classed as a retention cyst of the mammary gland from (unknown) obstruction of some of its distant tubules.

Dr. Tyson was inclined to consider that the tumor was a "retention cyst." *February 24th*, 1881.

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### 24. *Two cases of carcinoma mammæ.*

Presented by Dr. C. B. Nancrede.

The two cases I have to present for your consideration this evening have been announced to be of special interest because they illustrate the varied course pursued by different cases of the same disease. In brief, their histories are as follows:—

Case I. Mrs. C., aged 32 years, mother of four children, was seen by me in consultation with her attendant, Dr. G. A. Rex, September 3, 1880, when I found the following conditions present. At the lower inner part of her right mammary gland was a hard, circumscribed tumor, of the size of a walnut, which moved with the breast tolerably freely over the pectoral muscle. Under the anterior fold of the axilla was another mass, evidently consisting of enlarged glands, which fully equalled in size the mammary growth, if it did not exceed it in bulk. No tenderness over the tuberosity of the humerus was noted. The tumor had been noticed barely three months previously, which date must have corresponded almost to its incipiency, as the patient said that when she first detected it the growth did not exceed in size a small bean. A broken corset steel had bruised the breast at this point, and the injury had led her to examine the breast. The patient was a robust, healthy woman, and had been delivered only about six weeks when she detected the tumor. Her mother was healthy, but her grandmother had died with carcinoma mammæ. September 26, 1880, I removed the whole gland, with the superimposed skin, dissected off the pectoral fascia, fat, etc., and cleared the whole axilla of its glands, enlarged or not, until the capsule of the shoulder-joint could be readily felt. Antiseptic precautions were used, and the wound healed by the first intention, except the track of the drainage-tubes. The skin was so lax—owing to the recent cessation of lactation and the consequent shrinking of the breast—that, although the skin was extensively removed, very little tension resulted from the apposition of the cut surfaces. Over seven months after the operation the patient shows no return of the disease.

Case II. Miss H. R., aged 52, with a clear family history, came to me from a neighboring State on November 25, 1880, with her entire left breast converted into a growth of stony hardness. The growth did not much exceed in bulk the normal gland. The skin over the growth presented a roundish ulcerated surface about an inch and a half in diameter. This ulcer showed the characteristic appearances of carcinoma ulcerans. Between five and six years ago she first noticed the tumor, but in which segment of the gland I am unable to say. The growth slowly increased, with but moderate pain. Neither before nor after the operation could any enlargement of the axillary glands be detected. No tenderness over the upper extremity of the humerus could be discovered. November 29, 1880, the growth was removed, with the whole of the investing skin, part of the pectoral muscle, fascia, fat, etc. No glands could be found to remove. By dissection of the skin from the chest-walls, the enormous wound left was reduced to about one-third and dressed with carbolized oil. The patient did well, and when last heard of had no return of the trouble, although a small central spot, owing to tension, etc., had declined to heal, but *did not extend in the slightest*. Her health improved markedly after the operation.

It is hardly necessary to indicate the points of interest in these two cases, presenting as they do such a marked contrast as to the contamination of the neighboring lymphatics, and the importance of judging each case upon its own merits, and not upon any preconceived ideas as to when we should, or should not, operate.

Dr. S. W. Gross remarked that one point had been alluded to by Dr. Nancrede which he had been at some pains to test by his own experience, viz., the presence or absence of tenderness in the region of the greater tuberosity of the humerus in cases of carcinoma. Attention had been lately called to this by Mr. Snow, of London. In the last twenty cases of carcinoma mammæ under his care, careful examination had failed to discover tenderness over this portion of the humerus. *A priori*, such an involvement of the humerus is improbable, since it has been observed that this bone is very rarely affected in secondary cancerous disease of the osseous system. As his own experience in twenty cases totally failed to reveal such tenderness, and as the pathological fact just cited was against it, he was inclined to view the new symptom as worthless as a means of diagnosis.

Dr. Longstreth asked Dr. Gross whether any actual examination of the medulla of the bones had been made in the cases cited from an

English source which would prove this supposed involvement of the marrow-cells.

Dr. Gross, in reply, said that he was unaware of any such.

Dr. Longstreth said that he had asked the question because in one specimen examined by him he thought that he had found the medulla cells affected. He would like to ask the experience of the members as to whether the amount of cells, the signs of their active proliferation, and the involvement of the surrounding tissues could be relied on as to the rapidity of the return or the likelihood of metastasis, and related several cases where he had been induced by such appearances to predict a rapid recurrence, but where the course of the cases completely refuted his opinion.

Dr. S. W. Gross, in reply, said that if Dr. Longstreth continued to examine such cases and make similar prognoses he would find his opinions justified by rapid returns and dissemination in the majority of cases. In general terms, the less connective tissue, the greater malignancy.

Dr. Nancrede remarked that he would like to relate the ultimate result of a case of somewhat similar disease, which illustrated the extreme rapidity of course pursued by malignant growths where the cells were at a maximum. The members might perhaps recall a specimen exhibited by him before this Society, about a twelvemonth since, of large spindle-celled sarcoma of the breast, which had been opened by the attending physician in mistake for a chronic abscess, so deceptive was the sense of fluctuation produced by the excessive softness of the growth and the hemorrhages which had occurred in its substance. In removing it, Dr. Nancrede only succeeded in saving its pseudo-capsule and one or two small fragments for microscopic examination. In four months the lungs were crammed with deposits, which terminated this lady's life in about nine months after operation, and less than one year from the first appearance of the disease. *May 12th*, 1881.

## VI. THE NERVOUS SYSTEM.

### 1. *Two cases of syphilomata of the cervical dura mater.*

Presented by Dr. Charles K. Mills.

Syphilitic tumors of the spinal dura mater, and, indeed, spinal tumors of any kind, are sufficiently rare to give special interest to the specimens which I have the opportunity of presenting this evening. According to Rosenthal (*Clinical Treatise on Diseases of the Nervous System*, translated by L. Putzel, 1879, p. 238), an observation by him, in 1855, may be regarded as the first published example of a syphiloma of the spinal meninges, although other cases have since been reported by Potain, Macdowel, Wilks, E. Wagner, and others.

Case I.—W. H., aged 24, colored, married, for several years had been addicted to excessive venery, and twelve months before coming under observation had contracted a hard chancre. Two months after this he began to suffer with severe pain in the back of his neck, which soon became stiff and slightly twisted. These symptoms of irritation grew worse, and soon was added weakness, first of the arms, and later of the legs, with numbness and unpleasant sensations in the limbs. In six months he was bedridden.

On admission to the hospital, two months before his death, he was in a state of pitiful helplessness, bound hand and foot by disease. He was only able to sit up for a short time when fully supported. His neck was very rigid, and seemed to be shortened and sunken together. His head was held slightly bowed forward and twisted towards the right, any movement of it causing him great pain. He often felt as if he was being choked. He complained of dimness of vision. Both arms were much wasted, the legs, however, showed but little atrophy. He could not lift either hand from the bed, and could only bend the lower limbs slightly at the knees. The left side was possibly a little less helpless than the right. Electro-contractility was preserved. Sensation was diminished in both upper and both lower extremities, but was not mapped out. The slightest irritation of the skin would cause his legs to jerk violently. The thighs, apparently spontaneously,

would sometimes assume a semi-flexed position on the pelvis, from which he had no power to release them; they would have to be straightened by the attendants. His evacuations were involuntary, and incomplete priapism sometimes occurred. His sufferings could only be palliated. He continued to grow worse. Every night, and often during the day, he was attacked with severe pains in his knees and ankles. No acute bed-sores made their appearance, but several small ones from pressure formed at different points. On a number of occasions he had most distressing paroxysms. He would all at once be seized with a sense of choking and terrible dyspnœa, and would have to be propped and held up in bed; at the same time, profuse perspiration would break out. He finally died in one of these attacks.

The following was his temperature record for the two weeks preceding his death, the observations being made in the right axilla every morning and evening, the first temperature after the date being in each case for the morning:—

October 7, 97.6° F.; 97.4° F. Oct. 8, 98°; 99.6°. Oct. 9, 98°; 99°. Oct. 10, 97.9°; 98.5°. Oct. 11, 97°; 97°. Oct. 12, 97.7°; 97°. Oct. 13, 98°; 97°. Oct. 14, 100.5°; 100°. Oct. 15, 97°; 97°. Oct. 16, 97.5°; 98°. Oct. 17, 97.6°; 101°. Oct. 18, 97.4°; 98°. Oct. 19, 97°; 98.8°. Oct. 20, 99.4°. The average temperatures for the two weeks were, morning, 97.9°, evening, 98.3°.

Antisyphilitic remedies, rest, leeches, anodynes, and respiratory and cardiac stimulants were among the measures employed in the treatment of the case.

A post-mortem examination of the brain and spinal cord was made. The floor of the fourth ventricle, the corpora quadrigemina, and the choroid plexuses were apparently congested, being redder than usual; otherwise, the membranes and substance of the brain presented nothing abnormal.

On cutting through the vertebral column, a quantity of pus, and of pus mixed with blood, escaped from the spinal canal, particularly from the right side at the level of the third and fourth cervical vertebræ. The inner faces of the anterior segments of the second, third, and fourth cervical vertebræ were eroded and partly carious, the intervertebral cartilages being also partly destroyed by disease.

On the external surface of the dura mater, for a distance of two and a half inches in the upper and middle cervical region, was an exudation or growth, with an uneven, irregular surface, from one-fourth to one-third of an inch in greatest thickness; it was distributed so as to

girdle the cord, but was most developed in front. The inner surface of the membrane was smooth, and the cord within the limits of the diseased dura mater was flattened. The cervical nerves passing through the membranes were compressed and atrophied.

Nearly in the centre of the anterior aspect of the cord, about the level of the third cervical vertebra, was an oval or bean-shaped spot of bulging, half an inch long and one-eighth of an inch wide. It seemed to be defined from the surrounding tissue, looking like a small intramedullary tumor. The cord above and below it was softer than elsewhere. Three inches above the cauda equina was an exactly similar, but smaller, eminence.

Sections were made at intervals in the cord, and it, with the membranes and diseased mass, was examined microscopically by Dr. H. F. Formad, to whom I am indebted for the following report:—

"The thickening situated on the cervical portion of the dura mater was shown by the microscope to be an organized new growth, made up of embryonic connective tissue cells, those of round shape predominating, and containing also giant cells and bloodvessels with distinct walls. This structure, and the situation of the growth, in the presence of a specific history, indicate it to be a syphiloma (gumma). The adventitia of the bloodvessels of the dura mater was very much thickened, and in some places the walls of the vessels had a glistening homogeneous aspect, resembling that of amyloid degeneration.

"Transverse sections of the cervical portion of the spinal cord showed a total transverse sclerosis, the neuroglia being greatly increased, and the medullary sheath of the nerve-tubules nearly completely atrophied. The bloodvessels, which all had thickened walls, appeared to have undergone in many places amyloid degeneration, and in their course were seen numerous miliary hemorrhagic infarctions. Some of the latter were converted into minute abscesses, containing, besides pus, also compound granular cells and corpora amylacea. They sometimes reached the size of $\frac{1}{200}$ of an inch. The gray matter of the horns was comparatively less affected; some of the ganglionic cells were partly atrophied, while others appeared hypertrophied.

"The central spinal canal was seen to be filled with lymphoid cells imbedded in a reticulum of delicate fibres. I have met with such obliteration of this canal by cells several times, and it is probably of not rare occurrence in sclerosis; it is, however, described only once by Frommaun (*Untersuch über die norm. u. pathol. Anat. des Rückenmarks*, June, 1867).

"In sections made from the dorsal portion of the cord the sclerosis was only in the lateral and posterior columns; in the lumbar region, in the posterior only.

"The bean-shaped eminences, or areas of bulging, situated anteriorly in the cervical and lumbar cord, did not differ essentially in histological character from the structure of the cord itself in these locations, being composed entirely of connective-tissue elements."

Case II.—H. J., aged 31, colored, married, was admitted to the Philadelphia Hospital August 26, 1879. The history obtained from herself and friends was that in the preceding May she had been taken with a feeling of stiffness and pain in the neck. The pain radiated upwards to the head, and, to a less extent, down her back and arms; it was greatly aggravated by any kind of jarring or jolting; she suffered so much in this way that she soon became unable to ride in the cars. She lost flesh and became weak, but did not become distinctly paralyzed until two weeks before she was brought to the hospital. At this time, while attempting to dress herself, both arms suddenly fell powerless into her lap. The next day she could not stand, and since she had been completely helpless from the neck downwards.

On examining her, I found that she was compelled to lie constantly in one position on her back. She could not sit up at all, even with the amplest support. Her head and neck were held perfectly rigid, the face looking a little to the right, so that she rested on the right posterior portion of the head. Any attempt to change this position caused extreme pain, which shot upwards to the head. Both arms were entirely helpless. She was able to pull up the right leg so as slightly to flex it at the knee, but she could not do this with the left. She could perform movements with her feet, but with diminished facility. Roughly tested with the æsthesiometer, sensation seemed to be pretty good, but her answers in regard to the distance apart of the compass-points were uncertain and confusing. Reflex twitchings of the legs were produced when the points were brought in contact with the skin. The patella reflexes were present on both sides, but not exaggerated; the response on the right was poorer than on the left, the latter being about what is usually found in health. When first examined, on the day of admission, she had full control of her bladder and bowels. She had been troubled more or less for several weeks with shortness of breath, and three days before coming under observation she had had a severe paroxysm of respiratory difficulty, in which she thought she was "dying away." The attack was accompanied by profuse per-

spiration. Her pulse, recorded on the 26th, was 84, weak, but not intermittent.

Treatment was instituted, but without much hope, as the patient was, when admitted, evidently in the last stages of slow compression of the cervical cord. Her bed and pillows were so arranged as to give her head and body as complete rest as possible, and among the other measures adopted were leeches to the nape of the neck, potassium bromide and iodide in combination internally, hypodermic injections for the relief of pain, an ointment of belladonna, iodine, and mercury locally, and belladonna, digitalis, and ammonia for the paroxysms of dyspnœa and exhaustion. Fluid and semi-solid food was also frequently given. She grew worse from day to day; she was often bathed in profuse perspiration; dyspnœa and cardiac irregularity and weakness became greater, and she died, exhausted, August 31, five days after admission. Her temperatures, taken on the evening before her death, and on the day of her death, were as follows:—

| | Right axilla. | Left axilla. |
|---|---|---|
| August 30, . . . | 100° F. . . | 102.2° F. |
| " 31, . . . | 99.4° " . . | 99° " |

A post-mortem examination was made twenty-three hours after death. Post-mortem rigidity had not taken place to any marked extent, except in the fingers. No decided wasting of the body or limbs was present. The muscles of the neck were completely relaxed.

I carefully dissected away the extravertebral tissues,—muscle, fat, etc.,—to ascertain whether any disease outside of the vertebral column existed; but no evidence of such could be found,—no exudation, suppuration, or signs of inflammatory action. The spinal cord and membranes were examined in position after sawing through and removing the posterior portion of the vertebral column. On the left side of the cord, outside of the dura mater, a layer of dark, clotted blood could be seen, reaching upwards from the fifth cervical vertebra nearly to the upper limit of the cord. Externally, the upper portion of the dura mater for a distance of more than two inches was the seat of an irregularly-flattened exudation or growth, which was found on removal of the cord and membranes to be thickest to the left, in front and above. In the upper part of this diseased mass, at about the level of the second cervical vertebra, was a small dark clot about the size of a half-dime. The inner surface of the dura mater was smooth, but more opaque than usual. After removing the cord and membranes the cervical and dorsal

vertebræ were examined minutely, but nothing abnormal was found except a very slight roughening of the adjacent borders of the inner surfaces of the anterior segments of the second and third vertebral bones.

The upper cut end of the cord was softened and yellowish-white in color. The cord in the upper cervical region was more flattened than usual anteriorly.

The brain and its membranes were examined. The pia mater showed signs of recent hyperæmia, particularly about the junction of the ascending parietal convolution with the inferior parietal lobule. No other organs were examined.

A microscopical examination of the thickening of the external layer of the dura mater, made by Dr. H. F. Formad, showed it also to be a syphiloma, similar in structure to the specimen from the first case. Sections from the cord below showed likewise a transverse sclerosis.

*Remarks.*—Case I. is an example of an interesting compound condition: caries, probably syphilitic, of the vertebræ; syphiloma of the external layer of the dura mater; compression of the cord at the level of the tumor; sclerosis of the cord below the growth, at two points so marked as to appear almost like intra-medullary gliomata. In Case II. we have a syphiloma of the external layer of the dura mater, with compression of the cord and transverse sclerosis; and, in addition, dural hemorrhage, probably intercurrent with the growth. Disease of the bone was not present in this case. In regard to the first case, we have good authority for the view that syphilis may cause vertebral caries. The sclerosis of the cord below the tumor was doubtless of the nature of secondary degeneration.

During life I supposed that the first case might be one of external spinal pachy-meningitis, or spinal peri-pachy-meningitis, consecutive to, or in connection with, caries of the vertebræ. Such cases have been described by E. Wagner, Michaud, and others. They sometimes follow traumatism. Sometimes inflammations extend from the exterior through the intervertebral foramina.

Caries of the upper cervical vertebræ, as the specimens show, was, indeed, present. The probabilities are, however, as we have here an undoubted syphilitic thickening or gummy growth on the external layer of the dura mater, that the vertebral disease was also syphilitic.

In regard to etiology and symptomatology, my remarks upon these cases will be brief. In the first case, the patient had a clear history of chancre; in the second, although no history of syphilis was obtained,

the antecedents and surroundings of the patient were such as to make it probable that she had been infected. Both cases were striking illustrations of cervical paraplegia from compression-myelitis. This condition may arise in various ways, among which it is necessary to distinguish, if we would treat cases satisfactorily. It may be due to disease or injury of the vertebral column; to peri-meningeal, meningeal, or spinal growths; to wounds of the cord; to hemorrhage; to internal spinal pachy-meningitis; to disease affecting bilaterally the peripheral nerves; and to other causes.

In both cases the arms suffered first and most severely; in both sensation was impaired, but not to the same extent as motion. In the first case the tendinous reflexes were not examined, but skin reflexes were increased; in the second, the patellar reflex on one side was probably diminished, the other being about normal. The diminution in this case may have been due to the destruction by descending degeneration of the gray matter of the cord below the seat of the lesion. The tendency to priapism present in the man is of interest in reference to the question of the existence and position of a centre of erection in the cord. Several cases have been reported in which disease or injury of the cervical or upper dorsal region has given rise to priapism, it is supposed by some, from irritation of a spinal centre of erection. The respiratory paroxysms were among the most distressing manifestations. Cases of both cervical and dorsal disease often suffer from respiratory disturbances, and not infrequently, as in those now reported, die during such attacks, apparently from respiratory and cardiac paralysis. Occasionally, as in a case recently observed by me but not yet reported, asphyxia is produced by spasm of the muscles concerned in respiration. Usually profuse perspiration accompanies these seizures, probably showing implication of vaso-motor as well as respiratory nerve-paths and centres. The results of the temperature observations in the first case, although not remarkable, are worth recording. The temperatures generally and the average temperatures were below the normal. Wonderful oscillations of temperature have occurred as the result of spinal lesions, and particularly those of the cervical spine. *September 11th*, 1879.

2. *Tubercular meningitis, with a tubercle granulation on the floor of the fourth ventricle.*

Presented by Dr. Morris J. Lewis.

Mary O'R., æt. 6½ months, white, youngest of two children, eldest healthy. No history obtained of hereditary disease in either the father's or the mother's family. The father is said to have nasal catarrh and cough. The mother is a pale, thin woman; she has never had cough or night-sweats, but examination revealed harsh respiration both anteriorly and posteriorly at right apex, but no moist sounds were detected.

No history of constitutional syphilis could be elicited. Until the patient was four months old it remained to all appearances perfectly well; a slight nasal catarrh then made its appearance, and this was soon followed by a slight cough. The child was well nourished, her bowels were regular, and the passages natural in appearance.

On January 24, 1880, the child was first brought to the Children's Hospital Dispensary; she was seen by Dr. A. V. Meigs, and was suffering from nasal catarrh and slight cough. Shortly after this she was seen by me for the first time; the symptoms were as above described, and the child appeared to be doing well until the 18th of February, when there was a slight attack of vomiting, with conjugate deviation of the eyes to the right. These symptoms soon passed away, and no more serious ones appeared until March 1, when the mother noticed that the child did not appear to see; the hydrocephalic cry then made its appearance, and the child began again to vomit. During the night of March 2 she had three violent convulsions, each one lasting about fifteen minutes, and with about fifteen minutes' intermission; during these convulsions the right arm and leg and left side of the face were convulsed, while the left arm and leg were comparatively flaccid. The mouth was drawn strongly to the left, the eyes were turned upwards and to the left, and the head was retracted. The next day the child had slight diarrhœa, with green evacuations. On March 5 I visited the child at its home, and its condition was as follows:—

The child lay quietly in bed on its back, with its head retracted. The head was quite large, the anterior fontanelle very prominent and pulsating visibly, all the veins over the anterior part of the head large and tortuous; eyeballs not pressed downwards in the least; conjugate

deviation of the eyes left and upwards; left eyelid closed, right partly open, but this condition varied frequently; conjunctivæ hazy and injected, pupils dilated and immovable; child apparently blind, not noticing sunlight when flashed in eyes; skin of upper lip indurated and somewhat excoriated; toes of right foot powerfully extended, same true of left great toe; sensation apparently preserved on both sides, but a little better on right side; able to move both arms and legs, but right arm and right leg moved much more powerfully than those on the left side. Tongue coated with a dense white fur. Child has no teeth. Abdomen not retracted. Examination of chest and abdomen negative. Pulse 140, and temperature 99⅘° F. Cheyne-Stokes type of respiration well marked—14 per minute. *Tache méningitique* very slow in appearing, but quite pronounced, and lasting a considerable time. Commencing cyanosis.

The child was placed upon bromide of potassium and hydrate of chloral, of each three-quarters of a grain every three hours, and inunction of mercurial ointment twice daily.

Ophthalmoscopic examination revealed marked choking of the disk in both eyes, most marked in right. No tubercles detected in choroid.

On the following day the child was much the same; she had had no more well-marked convulsions, but the right arm and leg were kept in a constant state of motion, the knee being somewhat rigid. When disturbed or touched she moved the left arm and leg slightly, and when crying the mouth was drawn somewhat to the left.

In the evening, from six to nine, she had numerous slight convulsions, all of them confined to the right side of the body and the left side of the face. Numerous light-red blotches about a quarter of an inch in diameter were noticed on various parts of the body, sometimes disappearing soon after their appearance.

*March 8th.* Pulse 200; temperature 101⅖° Fahr. Cheyne-Stokes type of respiration still more marked; constant slight rigidity of right leg and arm; cried only when touched; utterly unable to nurse, but was perfectly able to swallow milk when placed in mouth; bowels not opened since the 6th; responded freely to injection; urinated about twice in twenty-four hours; conjunctivæ very much injected, and covered with purulent secretion; no conjugate deviation of eyes, but eyes were occasionally rolled from side to side; head still retracted. Applied blister to nape of neck.

*9th.* After passing a very restless night, with occasional slight spasms upon right side, she had a violent convulsion, which was de-

cidedly most marked upon the left side of the body, and shortly after this she died.

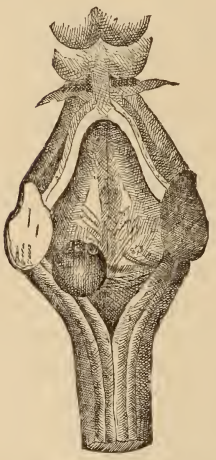
*Autopsy*, three hours after death, assisted by Dr. Louis Starr.

Child well nourished; rigor mortis commencing; skin mottled with large irregular light-purple spots, quite faint.

Head quite large, measuring 18 inches in circumference, and $10\frac{1}{4}$ inches from ear to ear across the top of the head; bones of the head freely movable upon one another; anterior fontanelle bulging. Upon opening skull there was seen a slight subarachnoid effusion; convolutions flattened; marked injection of all the vessels on convexity, very much more pronounced upon the right side; no tubercles detected.

Upon removing the brain a small amount of fluid escaped; pia mater at base very cloudy; convolutions on each side of the fissure of Sylvius upon the right side tightly adherent to one another, with some superimposed lymph; numerous miliary tubercles scattered on each side of fissure along its whole length, but very few noticed on left side of brain.

In the neighborhood of the paracentral lobule on right side a small patch of tubercles were seen; ventricles enormously dilated, especially the right one, and filled with clear serum, without lymph or detritus; ependyma perfectly normal in appearance; choroid plexus much injected upon right side, and with few miliary tubercles; brain very much softened; great ganglia at base normal; cerebellum apparently normal.



A pedunculated tumor, about the size of a large marrowfat pea, yellowish in color and soft in consistence, was seen springing from the left side of the fourth ventricle, just inside of the posterior pyramid, midway between the cerebellar peduncle and the calamus scriptorius; the tumor pointed upwards to the right, and a little backwards, touching the under surface of the middle lobe of the cerebellum; the tissue of brain at point of contact appeared somewhat reddened and softened. No miliary tubercles seen in neighborhood of tumor (see cut). The ocular ends of both optic nerves were markedly pyriform, and there was also choking of the disks. All the other organs normal in ap-

pearance except the left lung, which had three or four small, firm, white fibrous masses about the size of a grain of rice scattered through it. No miliary tubercles detected.

Thoracic and abdominal glands not enlarged. No tubercles seen elsewhere than in brain, and no cheesy focus discovered.

*Remarks.*—Several interesting points arise in the consideration of this case.

Huguenin (in *Ziemssen's Cyclopædia*) states that tubercular meningitis, unaccompanied by tubercular deposits in the other organs, or without a cheesy focus as a starting-point of the infection, but rarely occurs. In this case, it is true, no microscopic examination of the various organs was instituted, but, macroscopically, no tubercles could be detected elsewhere than in the brain, and those were plainly to be seen; and unless the slight degree of nasal catarrh can be taken as the starting-point of the disease, no focus could be detected, as the small, fibrous spots in the left lung were perfectly quiescent, and not the subject of any apparent retrograde change, nor were there any tubercles in their vicinity.

The nasal catarrh must be considered as the starting-point of the tuberculosis in this case,—a fact to be borne in mind as a possible termination of that obstinate but often considered trivial disease.

According to the statistics of Rilliet and Barthez, as given in Meigs and Pepper's work on children, only once in three hundred and twelve cases of tubercular meningitis were the tubercles confined to the meninges or brain. Eustace Smith, in his "Clinical Studies," states that acute tuberculosis of the pia mater may occur at any period of life, but is most frequently met with between the ages of three and ten years. It is, however, far from uncommon in infants a few months' old; but in such cases the distribution of the gray granulations is *almost invariably* general over the whole body, and the symptoms arising from the cranial cavity occur late in the disease.

Steiner makes the same statement in his work.

According to Huguenin, Rilliet and Barthez, and West, tubercular meningitis rarely occurs during the first year of life, the latter author having seen but five out of seventy-nine fatal cases where the diagnosis was verified by an autopsy.

The tumor, from its position, must have pressed more or less upon the important nuclei in the floor of the fourth ventricle, and this may account for some of the irregularity in the symptoms. It is to be regretted that no examination of the urine was made, as the proof of the

presence or absence of sugar would have been an additional point of interest, as the tumor so nearly encroached upon the point which, when irritated artificially, produces glycosuria.

The cross-convulsive movements, viz., right arm and leg and left side of face, lasting for several days, are difficult of explanation, as all the tubercular deposit was upon the right hemisphère of the brain, and these symptoms cannot be well ascribed to the tumor, at least according to our present scant knowledge of the functions of the medulla oblongata. The lesions in the present case are too general, however, for us to draw any conclusions as to cerebral localization.

The majority of the tumors of the medulla oblongata are tubercular in character, and are nearly always accompanied by intense hydrocephalus. The hydrocephalus in this case must be considered as partly due to the condition of the choroid plexus and to the tumor, which latter must have more or less filled up the fourth ventricle, and so have interfered with the return of the cerebro-spinal fluid. Indirect pressure may also have been made upon the venæ Galeni, and thus have aided in the ventricular dropsy. *March 11th*, 1880.

*Report of the Committee on Morbid Growths:—*

"A microscopical examination of a section of the tumor, situated on the floor of the fourth ventricle, shows its histological structure to consist of embryonal cells, separated by a very delicate reticulum of fibrous tissue,—adenoid tissue. At the periphery of the tumor this tissue is seen in an active stage of development; passing towards the centre, the cells gradually become more scattered and granular, the intercellular substance more distinct, until there is observed nothing but a granular débris, and, finally, in the centre, there is a loss of substance. The bloodvessels are seen with their lumina obliterated by cells and granular matter, which in transverse section give them the appearance of giant cells. The new formation is a tubercular granulation. *April 8th*, 1880."

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### 3. *Nerve-tumor.*

Presented by Dr. J. E. Garretson.

The egg-like tumor upon the plate was removed from the very centre of the posterior tibial nerve of a gentleman aged 55. The growth is of several years' standing; it is allied to a similar tumor in relation

with the outer cord of the lower cervical plexus. A tumor, supposed to be of like character, was dissected from the dorsal surface of the right index-finger. The growth shown had disparted the fibres of the nerve wherever it lay, and was the cause of much pain in parts supplied by the sacral plexus of nerves. The patient, himself a professional gentleman, had been treated a long time for rheumatism. The discovery of the tumor in the leg altered the treatment to that for neuroma. The growth removed from the finger is believed to have been an adenoma. The tumor upon the plate is reasonably to be designated a neuroma. It is presented to the Society for microscopical examination.

Dr. Seiler had examined the first growth removed by Dr. Garretson, and had found it to consist chiefly of bloodvessels whose endothelium was in a state of proliferation. The intervascular connective tissue was increased in amount. From such appearances he would class it as an angeioma. From a cursory examination of the section from the second tumor, now under the microscope, he would be inclined to consider it a spindle-celled sarcoma, but, to afford opportunity for a more thorough examination, he would ask for its reference to the Committee on Morbid Growths.

*Report of the Committee on Morbid Growths:—*

"A thin section made from the tumor situated upon the tibial nerve removed by Dr. Garretson shows, after staining with carmine, numerous nuclei, which are separated by a very indistinct and granular or slightly fibrillated intercellular substance. In many places the arrangement of the nuclei and cells is that which is observed when a section of very young tendon is examined,—longitudinal rows of cells placed at regular intervals and surrounding a bundle of fibrillæ; at other points transverse sections of the fibrillæ have been made, when the stellate lymph-spaces are seen anastomosing with one another. We were unable to find any structure which would indicate the presence of nerve-tissue. The growth may be said to consist of embryonal fibrous tissue."

*Oct.* 28*th*, 1880.

4. *Tuberculous growths (tyroma) of cerebellum.*

Exhibited by Dr. H. F. Formad.

This specimen and some notes of the history I obtained through the kindness of Dr. E. R. Girvin, who will present the interesting history of the case in full to the County Medical Society.

Miss G., aged 21, was never confined to bed with any sickness prior to the malady which terminated fatally. She complained, however, of occasional headache during the last eleven years, which she always located at the occipital region. She received an injury eleven years ago by a fall from a swing, striking this region of the head upon a projecting board, the accident being followed by loss of consciousness, which lasted, however, only a few minutes. With the exception of the periodical localized headache, which dated from the time of the injury, she remained perfectly well.

In February, 1880, the paroxysmal headache became of more grave nature, the attacks developed in frequency to one every five or six days, each attack lasting three to four days, the pain radiating over the entire top of the cerebrum, and attended with great hyperæsthesia. The skin cool; pulse compressible, thready, and irregular, ranging from 70 to 100. During April, May, and June the patient improved, but subsequently the attacks recurred with increased severity, and were followed by difficulty in vision, viz., double sight. Examination by Prof. W. F. Norris revealed choked disks affecting both eyes. Later, complete blindness ensued. There was a slight loss of power of left side of body; and sometimes twitching of the muscles of left side of face was noticed, and staggering gait. She complained sometimes of a momentary swinging or rotary sensation. The intellect was clear and the memory not impaired. During August and September the condition of the patient again improved. September 27th the attack suddenly became more severe, and, growing in intensity, terminated by death on October 1st. During the last few days of life there was loss of power of deglutition, also of sense of smell, taste, and hearing; general sensibility and clearness of mental faculty were perfect up to death.

A complete autopsy was objected to. I examined only the brain, with the following result:—

Skull normal. Dura mater normal, except over the left side of the

cerebellum, where it was thickened and adherent to the bone. Pia mater opaque, slightly thickened, and showing evidence of previous strong congestions and repeated effusions.

Examining the cerebellum by section, there were seen a number of yellowish-gray nodes of a new growth, varying in size from one-fourth to three-fourths of an inch in diameter, located both in the gray and white matter of the left side and at the middle of the cerebellum, in close proximity with, but not involving, the pia mater. The left side of the cerebellum was also thicker than the right. Over the seat of the tumefaction was a distinct localized septomeningitis, and adhesions between the membranes.

The cerebrum was very anæmic, otherwise of perfectly normal appearance in all parts. An increased quantity of fluid was noticed in the lateral ventricles.

*Microscopical examination.*—Nothing abnormal in cerebrum; the pia mater showed, however, numerous tubercle granulations in the course of the bloodvessels. Examination of new growth of the cerebellum proved it to be of a tuberculous nature, showing the structure of a non-vascular granulation-tissue containing numerous nodules, some of which were undergoing cheesy degeneration. Transverse sections of obliterated bloodvessels, and also true giant cells, were conspicuous. The granulations of the periphery of the nodes show a distinct attempt at fibrillation—the formation of a capsule. The location also of the new growths, consisting of such structural elements as described, leaves the diagnosis of *tyroma* beyond doubt, excluding two other possible new formations, the gumma and glioma.

The direct etiological relation of the new growth to the injury received eleven years prior to death is here highly probable, and hence in favor of the theory of the inflammatory origin of tumors.

*December 9th*, 1880.

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5. *Sections of the cerebral ganglia, with remarks on their anatomy and lesions.*

Presented by Dr. Charles K. Mills.

The specimens exhibited, nine in number, were (1) transverse and vertical sections; (2) longitudinal and vertical sections; and (3) horizontal sections of the ganglia. The preparations were not for micro-

scopical study, but were for the purpose of illustrating some points in connection with the coarse examination of the brain, with the view of accurately localizing and recording lesions. The following is an abstract of the remarks made by Dr. Mills:—

Although physicians seem lately to have awakened to the importance of carefully describing the limits of cortical lesions, they do not appear to be as fully alive to the equally important matter of definitely localizing lesions of the deep parts of the brain. By studying transverse, longitudinal, and horizontal sections of the cerebral hemispheres, one can soon obtain a clear idea of the size, shape, and normal appearances of the ganglia, and of the intervening and surrounding white matter. On the whole, perhaps, vertico-transverse sections will be the most available and lead to the least confusion in making post-mortem examinations. The profession has become familiar with the appearances presented by such sections from the plates in the works of Charcot, Ferrier, and others. The appearances of longitudinal and horizontal sections are not so well known. In the Annals of the Anatomical and Surgical Society of Brooklyn, for Jan. 1880, and in *Brain* for July, 1880, Prof. J. C. Dalton, of New York, has published papers on the *corpus striatum*, from a study of which many useful points can be learned with reference to longitudinal and horizontal sections. I show you sections similar to those figured in these articles, and will quote his remarks on horizontal sections: "In horizontal sections of the brain, the size and form of the cerebral ganglia vary greatly according to the level at which the section is made. At the level of the corpus callosum only a little of the arched portion of the corpus striatum is exposed, and none of the lenticular nucleus is visible. Somewhat below this, at the level of the fornix, the arched portion of the corpus striatum disappears, but there is a large oval section of the head in front, and a small one of the surcingle behind. Lower still the lenticular nucleus comes into view, separated from the head of the corpus striatum by the distinctly marked anterior prolongation of the internal capsule. At the level of the anterior commissure the lenticular nucleus and the head of the corpus striatum begin to fuse with each other, owing to the partial disappearance of the internal capsule between them. At this level the head of the corpus striatum is much reduced in size and altered in shape. The lenticular nucleus occupies a position outside and behind it; and still farther back is the section of the surcingle, which is here beginning to run downward and forward, along the roof of the inferior horn of the ventricle.

At a lower section still, the head of the corpus striatum would be united with the cortical convolutions in front of the Sylvian fissure, and the surcingle with the gray substance of the amygdala."

Attention may be called to a few points of special interest.

An erroneous idea of the exact position and relations of the ganglia may result from always studying brains after they have been removed from the skull. The superior surfaces of the intra-ventricular striate body and of the optic thalamus do not present a horizontal plane when the brain is in position in the skull, as they appear to do after it has been removed. Cutting into the lateral ventricles, with the brain still resting on the floor of the skull, it will be found that the faces of these ganglia are nearly perpendicular.

The slight depth of a large portion of the caudate nucleus might, perhaps, mislead in careless autopsies. At the head of this nucleus, where it partly blends with the lenticular body, the depth of gray matter is considerable, but it is slight as you go backward, ranging, in some measurements which I have made, from one-eighth to one-third of an inch.

When examining the lenticular nucleus, the difference in color of its different segments should be borne in mind. These are very marked, and, through ignorance of them, I have seen the error made of supposing that a normal lenticular ganglion was invaded by a large sclerotic nodule. On transverse sections in the anterior third a uniform grayish tint is presented, but in the posterior two-thirds, two, and sometimes three, colors or shades can be made out—one the grayish hue alluded to, and the others yellowish or yellowish-brown. On making a central longitudinal incision, the uniform gray tint of the heads both of the caudate and lenticular bodies is continued backward the whole length of the section, gradually tapering, however, and passing beneath the yellowish segments. These appearances are given by the two outer segments seen in the transverse cut. In infancy the differences in color between the parts of the lenticular nucleus are not so well marked as they are later in life.

We cannot be too particular in localizing lesions, and especially isolated lesions, of the ganglia and tracts. By so doing we may help to solve the problem—now so obscure—of the functions of these parts. The landmarks, as I have tried to show, being plain, this localization is not in reality a difficult matter. In some respects, indeed, it is less troublesome to locate and record lesions in the ganglia and capsules than in the convolutions, because in the latter we may have confusing

secondary and tertiary deviations from the usual forms. In considering isolated lesions it must not be forgotten that they may exert pressure on the surrounding regions.

According to Nothnagel (*Ziemssen's Cyclopædia*, vol. xii. p. 145), "lesions in the main cerebral ganglia from which spring the motor tracts of the crura cerebri, the *nucleus lenticularis* and the *corpus striatum* (*nucleus caudatus*), inasmuch as they are the most common of all, give rise to the most familiar group of symptoms." It seems to me, rather, that these lesions have taught us less that is exact than those of almost any other well-studied region of the brain, because, in the first place, absolutely isolated lesions of the ganglia and capsules are by no means common, and, secondly, because of the careless manner of making and recording lesions of these regions. It is probable that in the ganglia we have, in some way, representatives of each of the differentiated centres of the cortex. Certainly, as suggested by Charcot, the three gray nuclei of the lenticular body may be so many centres endowed with distinct properties and functions.

*Feb. 24th*, 1881.

## VII. ORGANS OF SPECIAL SENSE.

### 1. *Foreign bodies in the external auditory canal.*

Presented by Dr. Charles H. Burnett.

The young roach which I show you I removed alive from the ear of an Irish chambermaid in one of our large hotels last Tuesday morning. The patient stated that the evening previous, about nine o'clock, as she lay down upon her pillow, she felt something run into her ear; that she immediately experienced great and peculiar discomfort rather than pain, and that the intolerable annoyance of the movements of the creature and their attendant noises had continued all night, entirely banishing sleep. She looked very anxious and distressed, and had she not been of very large and muscular frame and in apparently robust health might have manifested graver nervous phenomena. I have known the entrance of a common fly into the ear of an infant to cause convulsions.

Under good illumination of the auditory canal by means of the forehead-mirror, I saw the central portion of the roach well down the canal, extending from above downward. This part I seized by means of delicate forceps with slender blades, and extracted the invader alive. It was 1 cm. long and 4 mm. wide at the widest part, not counting the antennæ, which are fully as long as it is. Instantly all the patient's discomfort ceased, and on examination it was seen that no harm had been done the ear at any point.

A great mistake is often made in such cases, first in supposing that the insect can do great harm, and secondly in making various rough and injurious endeavors at extraction.

The injury that does often arise in such cases is purely *post hoc*, and not *propter hoc*, as the aural surgeon sometimes has an opportunity of seeing.

Not long since, a distinguished aurist of Boston was called to see an old lady in one of the prominent families of that city, in order, as it was told him, to remove something from her ear. As he was hastily summoned, he went at once, and found the old lady in great distress

and just getting over the effects of ether. The statement made to him was that her little grandchild, while playing with her on her lap, had put a small shirt-button into its grandmother's ear, and that, though it produced no discomfort, it was deemed best to summon their family medical adviser, who was a homœopathic physician, and have it taken out. This was done, and the so-called physician examined the ear, and said he could see the "button distinctly, as it was quite pearl-like in appearance." He then proceeded to extract the foreign body, but failed to do so, and, besides, gave great pain to the patient.

Finally, ether was resorted to, and a prolonged gouging and probing carried on, but no button could be got from the ear. For various reasons the man ceased his manipulations and retired; the old lady recovered from her narcosis, but showed great signs of prostration; and, as it was supposed that the button was still in the ear, the aurist alluded to was summoned to extract it and relieve the suffering erroneously supposed to be due to its presence. He found that there was no button in the ear, and there was reason to believe there never had been, as the button which had been said to be in the ear was found subsequently on the floor near where the child and its grandparent had been sitting. What the first physician called in had seen and supposed was the button was the membrana tympani, which in the normal state looks like a pearl in color. To this he had directed his efforts, and *gouged it and the ossicles of hearing entirely out*, thus, of course, destroying the ear forever.

In my experience, a man, a junior partner in a machine-shop, called on me for relief from discomfort in his ear and deafness. He was a large, strong man, but was pale, anxious, and bathed in clammy sweat, manifesting, in fact, symptoms of shock. He stated that a few days previous, while crossing a street in front of a horse, the latter had splashed mud into his ear. No discomfort, save a little dulness of hearing, ensued; but on stating the occurrence to his comrades in the shop they insisted on his having the mud taken out, and they proposed to extract it, which they proceeded to do with wires and small tools. This caused great pain, but it was misconstrued as a sign of need of all the more sedulous endeavors at extracting the mud. Manipulation of the roughest kind was continued on the ear, until the man could endure it no longer. Great and continued pain ensued; the hearing remained dull,—in fact, became worse, and the man had to desist from work. Some little mud was taken out by his companions, and "several white pebbles," as he said (most probably the

ossicles). But as the pain and deafness continued, with some symptoms of dizziness and tension in the head, he concluded to seek medical advice.

Examination revealed *total destruction of the membrana tympani, it having been clearly cut away from its attachment to its bony ring, and all ordinarily visible parts of the ossicles had been similarly removed by his rough companions.* A few jets of warm water from a syringe would have removed all the mud had it been used at the first.

It is plain to be seen that two very important considerations arise at the outset when a foreign body is or is supposed to be in the ear. First, make sure of the diagnosis; and, secondly, make no effort at extraction which will do more harm than leaving the foreign body there, whether it be animate or inanimate. Presence of mind, too, on the part of the would-be surgeon as well as in the patient will always prove to be of the greatest aid.

This summer, a gentleman, just after he had gone to bed, felt an insect enter his left ear. He, of course, felt all the usual and peculiar annoyance from its movements. Being entirely alone in his house, he was thrown altogether on his own resources at the outset. He bethought himself in his extremity of a novel method of inducing the insect to come out, for he struck a light and held his ear as close as possible to it. He says he felt the insect turn and retrace its steps and come to the mouth of the ear, when he seized it with his finger-nail, which act caught and tore off its head. Death ensued, and all movements ceased in the insect, the body of which the patient removed at his leisure.

It would be well for doctors as well as patients to know that not the foreign substance so much as the unskilful and *rough treatment often practised is the real cause of the damage* which sometimes occurs *after* a foreign body has been in the ear. The most efficient treatment is to drop a little warm water or oil into the affected ear, and employ the syringe. Instruments should be used only by the expert.

*September 9th*, 1880.

## VIII. TUMORS NOT OTHERWISE CLASSIFIED.

### 1. *Tumor of submaxillary triangle.*

Presented by Dr. James E. Garretson.

The tumor presented was removed from between the leaves of the submaxillary triangle. The history associated with it is to be given in a few words. The patient from whose neck it was removed is a young gentleman, residing in Pittsburg, in this State. His age is 20 years. His general appearance indicates the existence of a dyscrasia. He is slow in his movements, pasty as to complexion.

I call the tumor by no name, because I know it alone as a growth that does not explain itself. To put it differently, it is a neoplasm as contradistinguished to several tumefactions of the region which it is easy to know all about and as easy to cure; inflammatory tumors, ranula, hygromas, for example.

The tumor, as you see, has been ablated. No assurance is possessed by me that it will not return; this, because I am ignorant of the meaning of its origin.

The growth is of four years' standing, and was first accidentally noticed by the patient as a kernel deeply seated in his neck. Enlargement has been gradual and regular.

Is the tumor upon the plate the submaxillary gland, or is it of other origin?

In removing the superficial leaf of the submaxillary triangles there are frequently met with two lymphatic glands lying directly upon the submaxillary. These glands I have on several occasions removed, finding the salivary organ intact. It is not uncommon, as my experience has demonstrated to me, to find that hypertrophy of these lymphatic bodies has caused a disappearance of the gland beneath, so that often it is the case that a submaxillary gland, described as being removed by the knife, had been taken away long before the day of operation through nature's process of absorption. The same thing has enlarged demonstration in the case of the parotid gland. I have myself done quite a number of ablations in this latter region, have

cleansed out the parotid space, have found the facial nerves imbedded in growths removed, have not seen a sign of anything like a gland-lobule remaining after my work was done, yet to-night am not at all assured that I have ever, in my life, removed the parotid gland.

The salivary glands are slow in yielding to malignant expression; adjacent glands, however, frequently enough take on neoplastic enlargement. Whether the body before us is the salivary gland, or one of the lymphatics referred to, it will be interesting to have determined by the microscope. Practically, however, the difference is nothing; the tumor upon the plate occupied the submaxillary triangle, and beside it there was no other body in that recess. The interest of the case lies in the question, Am I wrong or right in esteeming and treating the enlargement as a neoplasm? This, for the reason that if the tumor prove no neoplasm, the practice pursued exposes weakness. In position the growth possesses the hardness and common lobulation of scirrhoma. *October* 23*d*, 1879.

*Report of the Committee on Morbid Growths:—*

"The microscopical examination of the specimen presented by Dr. Garretson shows it to consist of a glandular structure made up of ducts and acini lined by epithelium. Also throughout the sections are seen numerous islands or patches, consisting of irregularly-shaped cells separated by a structureless hyaline intercellular substance. The formation would, therefore, be a cartilaginous development within a glandular organ. *November* 13*th*, 1879."

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### 2. *Tumor of the kidneys.*

Presented by Dr. C. Seiler.

The specimen presented is a piece of a tumor of the kidney which was sent to me for microscopical examination; and, as tumors of the kidney are rare, I thought it would be of interest to the Society, even though the history which I was able to obtain is very unsatisfactory.

The child from whom the specimen was removed was apparently healthy at birth, and remained so for eight months, when it began to sicken, and an enlargement of the abdomen was noticed, which continued to increase, until it assumed enormous proportions; the secretion of urine being, however, normal. The child died at the age of

13 months; and at the post-mortem examination three tumors were found in the abdomen—one in connection with the mesentery, and two larger ones, each of the size of a child's head, in connection with the kidneys. These seemed to be split by the tumors, and were very much enlarged.

A microscopic examination of the growth showed it to consist of dense connective tissue containing large spindle cells, which laterally showed, under high powers, transverse striæ. I therefore consider the tumor to be a rhabdo-myoma. *October 23d*, 1879.

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### 3. *Epithelioma of the lower lip.*

Presented by Dr. Morris Longstreth.

Dennis F. G., æt. 49, was admitted to the Pennsylvania Hospital, October 22, 1879, under the care of Dr. Thomas G. Morton. I am indebted to Dr. Baum, the resident physician, for the following history of the case. The man's history is good, as regards the occurrence of any malignant or hereditary disease in other members of the family. At the age of 10 he suffered from disease of the knee- and hip-joints; abscesses formed, and several large pieces of necrosed bone were removed from the femur, etc. Twenty years ago the man contracted a chancre, and since then he has had a large ulcer on the right leg. Nine years ago, while working in the sun, his lips, etc., were sunburned and parched, and subsequently the lower lip fissured in the middle line. The fissure became an open sore, or ulcer, and about four or five years ago an outgrowth commenced in the middle of the lower lip.

On admission, the growth was found to involve the entire lower lip, except the extreme margins at each angle; it measured two and one-half inches in length and about one and one-half inch in thickness. The surface of the growth was uneven and nodular, and covered with fungous granulation peculiarly exuberant in character. On the outer surface of the lip, near its inferior portion, there were numerous rounded, slightly prominent nodules, but not ulcerated on their surface. The hairs on this portion of the lip were strikingly rank in their growth, and were loose in their attachments, and easily plucked out from the skin. The lip, although very much everted, so as to allow the dribbling of saliva from the mouth, was found at the time of the operation firmly attached to the lower jaw.

The growth was removed by incisions carried vertically through the lip near its angles, and by a horizontal incision joining the two vertical cuts. The condition of the alveolar process of the bone—the incisor teeth being lost, and some granulation springing up from their sockets —suggested the possibility of the bone being involved in the growth, and it was deemed best to remove this portion of the jaw; the operation was done with Bonwill's engine, armed with a circular saw, and operated by Dr. Bonwill himself. The wound of operation was closed by a principal suture placed at the middle of each of the vertical incisions; the borders of the newly-formed lip were brought together by a series of sutures, bringing the mucous membrane in apposition with the skin, and thus covering in the raw surface. The removal of the bone allowed of a better closure of the wound. A small abscess subsequently formed under the chin, and was opened.

The microscopic examination, made from sections cut with the freezing microtome, showed the growth to be an epithelioma. As the physical characters of the growth indicated, the microscopic appearances presented by the specimens showed a marked hypertrophic enlargement of the papillary layer on the surface of the growth; in parts so marked was this development that the appearance presented closely resembled a simple papilloma. The tumor itself showed but very few "pearly" formations; the epithelial cylinders were very numerous in parts, and growing luxuriantly. The corpuscular elements were very abundant in the connective tissue surrounding the epithelial formations. The vascularity of the parts was greatly increased. The muscular fibres of the lip showed very marked atrophy, and in some parts had nearly disappeared. The bony tissue and its attachments were not involved in the growth. *Nov. 13th*, 1879.

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4. *Giant-celled sarcoma of the upper extremity of the humerus.*

Presented by Dr. Samuel W. Gross.

Mrs. J., æt. 23 years, fell on the ice on the 2d of January, 1879, but without any immediate ill effects. In July, or six months subsequently, she observed that the upper portion of the left arm was swollen, and that it soon became the seat of a dull, heavy pain, especially on motion, which has continued ever since.

On the 8th of November, when Professor Gross disarticulated at the shoulder by cutaneous flaps, the upper third of the humerus was converted into a smooth ovoid tumor, which measured one and one-quarter inch more in circumference than the opposite limb. Its consistence was elastic; its temperature was elevated one degree, but the skin, veins, and lymphatic glands were normal. The humerus was fractured, but the patient was not aware of that fact before she came under observation. After the overlying soft parts had been cleared away, the tumor, which was as large as a goose's egg, was found to be surrounded by a perfect periosteal capsule, which was continuous above with the thinned articular cartilage of the head of the humerus, and below with the periosteum of the shaft, the latter of which terminated abruptly in the midst of the neoplasm. Its tissue was of a uniformly grayish tint, friable, and had a fibrous tear.

Under the microscope the growth was found to be a typical giant-celled sarcoma, the very numerous multi-nucleated elements being contained in a spindle-celled matrix. *November 13th*, 1879.

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### 5. *Spindle-celled sarcoma over olecranon process.*

Presented by Dr. Samuel W. Gross.

A widow, æt. 61, came under my care at the hospital of the Jefferson Medical College, on the 20th of last August, on account of a tumor over the posterior face of the right elbow, which she stated was first observed two years previously as a moderately soft, painless nodule. Its further growth was slow, until June, 1879, or two months before she came to the clinic when it began to increase rapidly, and be the seat of a gnawing pain, which frequently interfered with her rest. On the 6th of August it began to ulcerate at its summit, and the suffering was aggravated; a week subsequently it bled rather freely. The wax model, made at the time, shows a conoidal tumor surmounting the olecranon process and the triceps tendon. The skin was of a bluish-red tint, pervaded by enlarged capillaries, and attenuated. At its base the neoplasm measured eleven inches in circumference, while it was three inches high, and its apex was ulcerated to the extent of an inch in diameter. In outline it was lobulated; its consistence was soft and elastic, and apparently fluctuating over the larger bones; and its temperature was three degrees higher than that of the opposite arm. The

veins in the immediate vicinity of the growth were more prominent than natural, but there was no lymphatic involvement.

The wet specimen shows that the tumor underwent marked changes during the three weeks and a half which elapsed between the date that the patient first came under my care and amputation. Thus, its circumference had increased two inches and its height three inches, while the ulcer measured three inches and one-half in diameter. The bones were more prominent, and the largest one fluctuated distinctly, and was evidently approaching ulceration.

Amputation of the arm was practised at the junction of its middle with its lower third, on the 20th of September, and the patient was discharged at the expiration of five weeks.

Section of the tumor and the underlying structures, which was attended with the escape of straw-colored fluid, disclosed that it was composed for the most part of a succulent, striated, yellowish-white tissue, with here and there gelatinous areas and a few small cysts. The older portion, or that which lay nearest to the olecranon process, was deeply blood-stained. The triceps tendon was infiltrated by the morbid deposit. The ulna was not involved, the tumor, which developed in the subcutaneous connective tissue, being merely attached to its investing periosteum.

On microscopical examination the growth was found to be a hemorrhagic large spindle-celled sarcoma, with an occasional giant element.

*November* 13*th*, 1879.

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### 6. *Case of tumor of scapula.*

Presented by Dr. Nancrede.

Isaac L., æt. 23 years, was admitted on September 24th, 1879, to the men's surgical ward of the Protestant Episcopal Hospital with a large tumor of the scapula. He was a farmer by occupation, had been healthy, and his family history was good in every respect. For about a year past he had been troubled by occasional cough, which was relieved by simple expectorants. About one year ago, without any assignable cause, a small tumor of the size of a hickory-nut was noticed growing from the left scapular region, in the neighborhood of the spine of that bone. It rapidly increased in size, until at the time of his admission it measured about six inches in both the transverse and

vertical diameters. No special pain was complained of, and his appearance apparently indicated robust health, although, perhaps, he was somewhat paler than a man of his type should have been.

The growth seemed firmly adherent to the scapular spine, and, being bound down by the dense infra-spinatus fascia, appeared to involve the bone still more extensively. When the removal of the growth, however, was attempted, upon September 26th, 1879, it was found merely to have sprung from the lower border of the spine of the scapula, no other portions being involved, and not the slightest trace of infiltration was observed. A portion of the flaps sloughed,—probably from pressure used to arrest consecutive hemorrhage,—and the wound did not heal well, suppurating freely; but the discharge did not become offensive until towards the close of life. Two weeks before the fatal termination, he complained of pain over the left side of the thorax, which, however, was quickly relieved by treatment. Following upon this, exhaustion became marked, necessitating the recumbent posture, and was accompanied by profuse sweating, chiefly affecting the head, back, and thorax. There was also some muco-purulent expectoration, lasting, however, but two or three days.

Physical exploration revealed impaired resonance of both lungs, specially the right, over which mucous and sonorous râles were heard. The respirations varied from 18 to 32. Steadily increasing and marked dyspnœa was rapidly developed, as if from pressure on the bronchial tubes. The expectoration was chiefly frothy mucus. Death occurred on November 6th, 1879.

*Sectio cadaveris.*—This revealed numerous tumors occupying the substance of both lungs, chiefly the right, which was almost wholly converted into a mass of sarcomatous tissue. The thoracic walls were also studded with similar growths. The right lung was adherent to the diaphragm, and from the thoracic cavity of the same side five and a half quarts of sanguineo-serous fluid were removed. The liver and kidneys were both healthy. Other organs not removed.

For the notes of this case Dr. Nancrede said that he was indebted to Dr. Bickford, who had been his resident at the time of the man's admission, and who attended him until his demise.

*January 8th*, 1880.

*Report of the Committee on Morbid Growths:—*

"The tumor presented by Dr. Nancrede from a microscopic examination is found to have the histological structure of a sarcoma, viz.,

consisting of cells possessing a relatively large nucleus; the majority of the cells have a spindle-shape. The intercellular substance is small in amount and granular. The bloodvessels present the characteristic appearances met with in this class of neoplasms, being embryonal in structure. From an examination of a large section, it is seen that the growth has penetrated between the neighboring fasciculi of muscular and fibrous tissue, and that the surrounding adipose tissue is also infiltrated by the new formation. *January 22d*, 1880."

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7. *Carcinoma recurring in the axillary glands after excision of a tubular carcinoma of the male mamma.*

Presented by Dr. S. W. Gross.

On the 24th of March, 1879,[1] I exhibited a scirrhous carcinoma of the breast of twelve months' duration, along with two infected lymphatic glands, which had been removed from a male subject, 59 years of age. On the 7th of the following August, the patient returned to me with a cluster of glands in the axilla, which were hard, discrete, and mobile. The cicatrices of the former operation were sound, and the skin was free from disease. I advised immediate operation, but this was postponed until December 31, when I excised, along with a portion of the adherent skin, a mass as large as a hen's egg, which was composed of twelve closely united glands, as well as four isolated glands. They were for the most part densely hard, although five were elastic and deeply blood-stained. Their structure, under the microscope, reproduced the characteristics of the primary growth.

*January 8th*, 1880.

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8. *Case of large spindle-celled sarcoma of mammary gland.*

Presented by Dr. C. B. Nancrede.

Mrs. F., æt. 35 years, the mother of four children, first noticed, accidentally, a small "lump," the size of a hazel-nut, in her left breast about two months before I saw her. It grew rapidly, but painlessly,

[1] See Phila. Med. Times, vol. ix., No. 303, p. 484.

for about six weeks, at the end of that time having reached the size of a doubled fist. About two weeks before I saw her there was a sudden and rapid increase of the tumor, attended with considerable pain. When she first noticed the "lump" her physician was consulted, who, without examining the growth, prescribed belladonna and iodine ointment as a local application. Two weeks before the operation, that is to say, about six weeks after first noticing the tumor, pain compelled her to submit to an examination by her family physician, who discovered, as he thought, obscure fluctuation. At a spot about two inches from the nipple, at the upper outer portion of the breast, the skin was red, inflamed, thinned, and presented appearances of pointing. After poulticing for some days her physician put in an exploring needle, and found what he thought was bloody pus. The following day he opened the softened spot by an incision, giving vent, however, only to about an ounce of grumous blood and clots, which microscopically revealed nothing special. No marked diminution in size followed the incision, although relief was experienced from the reduction of tension. Poulticing was continued, but no pus, only bloody oozing, accompanied by the extrusion of small clots, followed these applications.

I was asked to see the case in consultation, when I found a growth of a smooth hemispherical form occupying the whole breast. It was elastic to the feel, and had an obscure sense of what might be mistaken for fluctuation. A probe introduced through the incision passed for several inches in every direction through a mass of the consistence of currant-jelly. The passage of the probe was followed by considerable bloody oozing. No adhesions to the deeper parts existed, and there were no glandular enlargements.

On January 28, 1880, I removed the breast, with considerable difficulty, owing to the incision in the tumor, which permitted the diffluent contents to flow away, so that by the time the operation was completed little beyond an empty cyst remained. The contents resembled semi-clotted blood, and certainly but few fragments were detected which to an inexperienced eye were aught else than blood-clot.

A microscopic examination sustained the opinion given at the operation that it was a rapidly-growing sarcoma, into which hemorrhage had taken place. It consisted mainly of large spindle cells, with here and there the remains of proliferating gland-structure, all being more or less infiltrated with blood.

The case is of interest owing to the risk of confounding an exceed-

ingly malignant growth with an abscess before opening, or afterwards with a "blood tumor," which, much to the surprise of the surgeon, rapidly returns after removal. *February 12th*, 1880.

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9. *Intra-ocular tumor.*

Presented by Dr. C. B. Nancrede, from Dr. Albert G. Heyl.

The patient, Miss C., æt. 48, from whom this specimen was taken, applied for treatment at the eye and ear department of the Episcopal Hospital, February 16, 1880. The clinical history is as follows:—

About one year ago the patient noticed that the sight of the left eye was affected; she was not aware of any pre-existing inflammation in the eye, or of any injury to the organ. About three months ago a violent attack of inflammation of the eye occurred, characterized by great pain, exophthalmos, injection of the conjunctiva; for this she was treated antiphlogistically by Dr. J. R. Weaver, of Norristown, Pa. The acute symptoms subsided, but the eye ever since has been more or less irritable, and at times painful. At Dr. Weaver's suggestion, she finally consulted me. The examination of the eye resulted as follows:—

R. E., V= $\frac{20}{20}$. No abnormal appearances in the fundus.

L. E., entire loss of sight. Eyeball shrunken, presenting the condition known as phthisis bulbi. The conjunctiva somewhat injected. The sclerotic dimpled in two places, near the ciliary region, in the upper inner quadrant, as though bands of connective tissue in the interior of the ball had dragged upon it. Ball tender to touch at one or two points, but not generally sensitive. Anterior chamber shallow; pupil dilated; lens completely cataractous; at the position of the anterior pole, and within the capsule, a small black body about the size and shape of a canary-seed.

The removal of the eye was advised, and accomplished on February 17. The enucleation was attended with a little difficulty, owing to the matting together of the tissues about the insertion of the muscles.

After removal of the eyeball, the cornea, which was found to be greatly thickened, was cut away, in order to examine under the microscope the black spot in the lens.

The sclerotic, in addition to the two indentations already spoken of, had also a deep sulcus near the equator bulbi, apparently due to the

retraction of the interior structures. An equatorial cut was made through the sclerotic. It was then found that the interior of the eye was composed of a tough, blackish mass, between which and the sclerotic a cavity existed, apparently due to the contraction which this interior mass had undergone. This mass was adherent about the optic disk; possibly also in the ciliary region, but to a very slight extent, as the anterior half of the sclerotic separated from the interior mass with very little traction. The depression in which the lens lay was perfect in shape; the posterior capsule could be peeled from it. The lens was of the consistence of very thick gruel.

*February 26th*, 1880.

*Report of the Committee on Morbid Growths:—*

"A thin section of the new formation developed in the eye, upon microscopic examination, is seen to be made up entirely of spindle-shaped cells, the protoplasm of which is densely pigmented with minute granules of black pigment. The bloodvessels are embryonic in character. The growth is a spindle-celled melanotic sarcoma.

*March 25th*, 1880."

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10. *Dermoid cyst of left cornea.*

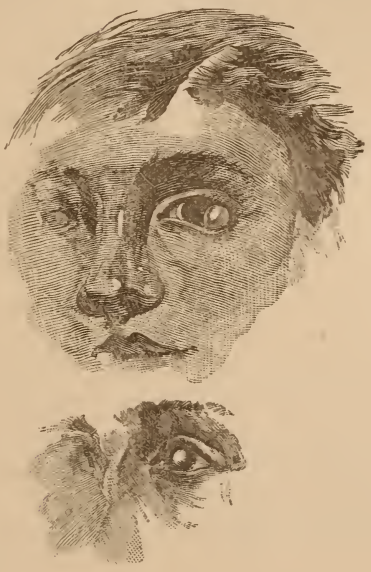
Presented by Dr. S. D. Risley.

A. R., æt. 18, consulted me, January 20th, 1880, regarding a tumor on his left eye. The following history was given. His mother noticed, during very early infancy, a white spot, the size of a large pin's head, near the external limbus of the cornea, situated entirely on the sclerotica. It gradually increased in size as the child grew. It has never caused any pain or trouble of any kind, other than the defective vision.

*Present State.*—O. D. healthy; V=$\frac{6}{6}$. In O. S. deciphered Sn. c.c. at 1 M.

There is on O. S. a pinkish-white tumor five millimetres in height, eight millimetres in vertical diameter at the base, and one centimetre in its horizontal diameter. In shape it is a truncated cone. It is vascular at its base, and has four black hairs, the length of the cilia, growing from the top, with numerous finer, colorless ones, seen only with lens. The base horizontal line is situated half on the cornea,

half on the sclerotica, and is two millimetres below the horizontal meridian of the cornea. The corneal side of the cone is steeper than the scleral side, and the base more sharply defined. The cornea is hazy, one millimetre and a half around the base, and is lifted up above



Dermoid Cyst of Left Cornea.

the surrounding cornea, which is elsewhere transparent. The iris reacts promptly, and the anterior chamber is perfect. The deeper media are transparent, and the fundus healthy.

The accompanying sketch very perfectly represents the appearance of the growth, which is undoubtedly a dermoid cyst.

*March 25th*, 1880.

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11. *Spindle-celled sarcoma of the lower jaw.*

Presented by Dr. J. Ewing Mears.

The specimen involved the greater portion of the body of the lower jaw, which was removed by operation from a boy 11 years of age. Ten months before the operation for the removal was performed, the boy was struck upon the jaw over the region of the left canine tooth,

which was carious; soon after swelling appeared at this point, and continued until the growth reached the size to be observed in the specimen. Including the bone, which it embraces, the tumor is the size of a large lemon, projecting more from the external surface than from the internal, and extending laterally from the second bicuspid tooth of the left side to the first bicuspid tooth of the right side. On the left side the teeth were quite loose, being pushed from the alveoli and held by the soft structure of the tumor. On the right they were firmer, and not dislodged to the same extent as those on the left. Examination shows the tumor to have taken origin in the periosteum, and to have involved this membrane in its growth to the extent described. Microscopic examination reveals its structure to be that of a sarcoma of the spindle-celled variety. The growth was removed with the portion of the jaw involved, by making an incision along the inferior border of the bone from just in front of the anterior inferior angle of the masseter muscle on one side to the corresponding point on the other, beginning and ending at these points, so as to avoid wounding the facial arteries. A short vertical incision from the symphysis of the jaw to within *one inch* of the *border* of the *lower lip* was also made, and the flaps dissected upward, in this way fully exposing the tumor. The muscular attachments, with the mucous membranes, were divided, and the bone sawn through on either side. The patient made a good recovery. *June 24th*, 1880.

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12. *Necrosis of superior maxillary bone following typhoid fever.*

Presented by Dr. J. Ewing Mears.

This specimen of necrosis of the superior maxillary bone was removed by operation from a boy six years of age, who had suffered from an attack of typhoid fever. The history given by the mother of the patient was as follows: "Fifteen weeks since, the boy was attacked with typhoid fever, and was quite ill for three weeks. During the fourth week of the disease the right side of the face gradually swelled, and, under the belief that an abscess was forming, a poultice was applied. Suppuration ensued, and a copious discharge of very offensive pus escaped. The teeth, as far forward as the canine, became loose, and were removed." The soft tissues gradually receded from the necrosed portion of bone, exposing the alveolar border. Although

the dead bone felt quite firm in place, it was thought best to remove it, believing that it was detached, and was held by the soft structures. These were accordingly separated by the knife, and the piece was grasped by the bone-forceps and dislodged. It measured one inch and a quarter from before backwards, and one inch from below upwards, and consisted of the greater portion of the body and alveolar process of the bone, with parts of the malar, nasal, and palatine processes.

The occurrence of necrosis of the jaws after attacks of the exanthematous diseases, such as smallpox, scarlet fever, and measles, is well established by the numerous instances which have been recorded, but is regarded by Mr. Salter (*Holmes's Surgery*) as rare after the continued fevers. Dr. Keen (*Toner Lectures*, 1877) also alludes to its rare occurrence *after* typhoid fever. *June 24th*, 1880.

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13. *Fibroma from the palmar surface of the hand, apparent exciting cause of reflex (Jacksonian) epilepsy.*

Presented by Dr. Charles K. Mills.

I am indebted to Dr. L. F. Flick for the opportunity of presenting this specimen, the patient from whom it was removed having been for a long time under his care.

M. S., æt. 15, when four years old, fell down-stairs from a height of five steps, striking on the right palm. The hand was not bruised or cut, but the child suffered great pain in it for several weeks. Six months after the fall, two little growths about the size of a pea made their appearance, one on the palmar surface of the wrist, over the position of the common tendon of the flexor sublimis digitorum, the other in the palm of the hand, over the tendon going to the index-finger, about one-third of an inch from the metacarpo-phalangeal joint. The tumor at the wrist was painful, and grew gradually for five years until it became as large as a hickory-nut. It was then treated systematically with liniments and bandaging, and in three years more disappeared. The tumor in the palm continued very slowly to increase.

Six months after the first appearance of the growths the child began to suffer from attacks of spasm, at first affecting chiefly the right hand. They recurred usually once in about ten days or two weeks. The spasms gradually grew worse; eventually the right arm and leg and the right side of the face would jerk. For the last ten years she

has continued to have seizures, generally at intervals of a week or two, but sometimes oftener. The limbs and face of the right side have always been more affected by the spasm than the same regions on the left. The twitching has invariably begun in the fingers of the right hand, and has been preceded by pain in the palmar tumor. The patient, who has always remained conscious, as the attacks have come on would grasp her right hand with her left, or would call on some one else to do this. Chloral, bromides, and other antispasmodics have been used freely, but without any evident effect.

In February, 1880, she began to have a series of paroxysms much more violent than any she had before experienced. At first the attacks came only more frequently during the night, especially after twelve o'clock. After a few days, however, they increased in number during the day also, until finally they recurred every two or three minutes day and night, ether, bromides, and other medicines having no effect. Menstruation now set in for the first time, and at once the convulsions ceased, after having continued as just described for two weeks. She grew stronger speedily, and for four months did not have another seizure. She now again began to have occasional fits. Menstruation was not well established.

Early in September, 1880, a second continuous series of convulsive attacks began. At this time, in addition to her dysmenorrhœal symptoms, her digestion was much deranged. I saw her first, with Dr. Flick, on the 15th of September, and since have seen her frequently both during and between her paroxysms. A curious feature has been that as soon as she would fall asleep an attack would come on, while during her waking moments she would often rest easy. She could sometimes control the spasms by force of will; and a smart slap with the hand would sometimes cause the attack to cease.

The following is a description of the usual character of the attacks, as determined from the notes of Dr. Flick, and from personal observation :—

The distal phalanx of the ring-finger of the right hand was first flexed; secondly, a few spasmodic movements of flexion would occur in this finger; thirdly, the other fingers and thumb of this hand would begin to twitch convulsively—the second phalanges would be flexed, the last extended; fourthly, the clonic convulsive movement would extend the right hand, forearm, and arm, and simultaneously the muscles of the lower part of the right side of the face would become

affected with spasm, a tremor also appearing in the tongue during this period; fifthly, the right arm and leg would now become affected with a clonic spasm, causing them to assume positions of flexion, the head, neck, and body being drawn by the spasm, at the same time, to the right, a condition of pleurothotonus being, in fact, produced. The seizures would pass off with a very severe jerking movement of the right shoulder, and a renewal of the twitching of the muscles of the right angle of the mouth. These movements of the shoulder and mouth would sometimes occur only once, just before the close of the attack; more frequently, however, they would take place two or three times in succession. Occasionally the patient would bite her tongue during the paroxysm. She apparently was never entirely unconscious during an attack, no matter how severe it might be. During the height of the convulsion, if her hand was pressed too hard, she would manage to gasp out, "Don't!" or to make some other exclamation.

After trying bromides, morphia, etherization, blistering, etc., without avail, I advised the immediate removal of the tumor. On the 26th of September, Dr. S. W. Gross removed the growth, Drs. Hearn, Kirkpatrick, Flick, and myself being present at the operation. The tumor was found to be a firm, lobulated mass, three-fourths of an inch long and about half an inch in each of its other dimensions. When it was first exposed, a small nerve-branch could be seen passing over its upper inner edge. It was somewhat firmly attached to that tendon of the flexor sublimis digitorum which goes to the index-finger.

The growth was placed for microscopical examination in the hands of Dr. Carl Seiler, who reported it to be a fibroma.

The following, in brief, is the history of the case for the four weeks which have elapsed since the operation. She has been kept absolutely without treatment for the spasms. She improved at once in her general condition. She has averaged a little less than one attack a day since. The right hand or arm has never been affected in any of these seizures, only a few of which have been severe.

*Remarks.*—I am inclined to look upon the spasms in this case as primarily, at least, reflex, the fibroma of the palm acting as an exciting cause. This seems probable for a number of reasons. (1) The spasms appeared after the fall and the development of the growths. (2) Before the removal of the tumor the spasm invariably began in the finger of the right hand, on the palm of which the growth was situated. (3) The spasms were usually of the nature of monospasms, or unilateral convulsions of the right side of the body. (4) The

spasms became much less frequent and severe, and ceased to begin in the fingers of the right hand, after the operation.

The convulsive disease from which this child has suffered has all the characteristics of what has been termed "Jacksonian epilepsy," in honor of Hughlings-Jackson. By a "Jacksonian epilepsy" is meant a convulsive affection confined to a group of muscles, to a limb or to two limbs, or to face and limbs of the same side, and usually commencing in the same way. Consciousness sometimes is and sometimes is not lost. Unilateral convulsions are more likely to be associated with loss of consciousness than spasm confined to the face or to one limb.

Have we in a case like this any real morbid changes in the brain? Without doubt, I think, such changes have taken place. Post-mortem examination would probably reveal inflammatory changes of the cortex, most marked in the middle Rolandic region of the hemisphere. The presence of the tumor in the hand may, of course, be merely a coincidence, and we may have here in the arm-centre of the left side a tumor, cyst, tubercle, or other lesion, irritative in character, and the primitive cause of the monospasm. Cases are on record corresponding in their main features, as regards the origin and progress of the spasmodic manifestations, with our case, and in which, after death, small cysts or tumors have been found in the motor zone of the convolutions. *October 28th*, 1880.

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### 14. *Epithelioma of the foot.*

Presented by Dr. H. F. Formad.

This specimen is taken from the dorsum of a foot removed by Professor Agnew last winter in the University Hospital surgical clinic. The following history was obtained through the kindness of J. W. Worrell, student of medicine at the University.

The patient, a robust young man of 27 years, fourteen years ago had a compound fracture of the os calcis, the foot being caught between the wheels of a threshing-machine. The patient recovered from the injury with a stiff ankle-joint, but at the end of six months the cicatrix began to slough, in which condition it remained for six and a half years; then it again healed up entirely. The cicatrix, which had a very scaly appearance, remained, however, closed but one month; then it opened by one single long fissure or break, and has re-

mained an open superficial ulcer ever since. For five years the edges of the ulcer remained smooth, then they became rough. The ulcer increased very slowly in size at first, but extended more rapidly during the last six months. There has always been a very offensive discharge; the sore has always been painful and itching; never bleeding of itself, but bleeding readily on the slightest injury. At the time when the patient came to the University Hospital clinic the ulcer covered an area of twenty square inches, in its longer diameter reaching around the dorsum of the foot, from the external to the internal malleolus. The diagnosis was, as in all such cases, that of indolent ulcer.

Microscopic examination of sections taken from all parts of the ulcer revealed it to be a typical squamous epithelioma crowded with characteristic pearly bodies.

Dr. J. H. Packard agreed with Dr. Formad in thinking that certain indolent ulcers were in reality epitheliomatous, and instanced a case in point.

Dr. Formad said that he should like to call attention to the youth of the patient from whom this specimen had been removed, for the books generally state the contrary. In his experience he had had several opportunities of examining similar ulcer-like epitheliomata removed from comparatively young subjects.

Dr. Nancrede said that some five years ago he had presented to the Society for Dr. J. Ashhurst, Jr., a specimen of pigmented papilloma of the groin, which showed the commencing invasion of the subcutaneous tissue by epithelial elements. The patient from whom it was removed was about 18 years of age, and she had remained perfectly free from the disease up to the present time, he believed, owing to the early and free removal of the growth. He also cited a case of so-called "Marjolin's ulcer," giving a detailed account of the microscopical appearances.

Dr. Tyson agreed with the views propounded by Dr. Nancrede, and said that the evident inference to be drawn from the facts related this evening was that early and radical surgical intervention should be resorted to in such cases.

Dr. Bruen said that he knew of numerous cases of ulcers which had proved intractable for many years—perhaps twenty—and which yet showed no evidence of epitheliomatous change.

Dr. Formad could not admit that the base of an ulcer, consisting as it does of mere granulation tissue, could become epitheliomatous. The

indurated margins of the ulcer, containing epithelial elements, could, however, readily undergo this change.

Dr. Tyson said that Dr. Formad's remarks really opened up the much-vexed question of epithelial new formations. Where a new islet of cicatrization appears in the centre of a sore, the same difficulty arises as to the explanation of its genesis.

Dr. Packard said that his remarks had reference to mistakes in diagnosis.

Dr. Shakespeare remarked that he had lately been asked to examine a fragment removed from an obstinate chronic fungous ulcer on an old woman's leg, who had been for many years an inmate of Blockley. Under the microscope, sections of this showed plainly its epitheliomatous nature. *Nov. 11th*, 1880.

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### 15. *Spindle-celled sarcoma of the skin*

Presented by Dr. S. W. Gross.

Records of examples of sarcoma of the skin in the medical journals and references to that disease in the text-books are so very uncommon that I am induced to bring the specimen before the Society.

A married woman, 23 years of age, was brought to my clinic at the Jefferson Medical College Hospital on the 29th of September, 1880, on account of a painless tumor of the integument of the buttock, which was of the size of a small lemon, of a firm elastic consistence, and with a discolored and rugous surface. She first noticed it as a shot-like tubercle about three years previously, and she informed me that she had struck it a few weeks before, and that the injury was followed by slight bleeding and rapidity of growth.

On section after removal, the cut surfaces were of a rosaceous-yellow shining hue, and had a fibrous tear. Microscopically, the growth was characterized, as can be seen from the accompanying slide, by small spindle cells contained in a homogeneous intercellular substance. Up to the present time the tumor has not recurred.

Dr. Duhring asked whether in this case there was more than one growth. Although it was now, of course, difficult, or impossible, to determine the exact starting-point of the neoplasm, yet he was inclined to think that its site had been the subcutaneous cellular tissue. Dr. Gross, he remarked, was correct as to the rarity of such growths,

all authorities making similar statements. Indeed, Kaposi, the most recent German writer, one who has devoted especial attention to the pathology of skin affections, only devotes a short chapter in his treatise to sarcoma of the skin. He treats of the disseminated pigmented form only, which seems to be the most usual multiple form assumed by sarcoma when attacking the integument. Dr. Duhring knew of only one instance which had been reported in this country within the past ten years. This variety of sarcoma, however, was of such a different form from that presented this evening that he would not enter into any further details.

The whole subject of sarcoma of the skin had been opened up by a case reported by him. He had described the case at the meeting of the American Dermatological Association held at Saratoga in 1878, as an "Inflammatory Fungoid Neoplasm." A year later, at the third meeting of the same Association, he had read a supplemental report upon the case, when, owing to the report of Dr. Heitzmann, he had been inclined to suspect its sarcomatous nature. The disease represented by this case was closely allied to sarcoma, if not true sarcoma. About the same time Geber reported a similar case, also under the caption "Inflammatory Fungoid Neoplasm."

Kaposi, in commenting recently on these cases, says that he has no doubt of their sarcomatous nature. All the cases so far have proved fatal in about two or three years. Dr. Gross had described his case as a "spindle-celled" sarcoma. Dr. Duhring would like to ask whether, in his experience, the round-celled was not much the commoner variety.

Dr. Gross said that he had notes of eight cases of sarcoma of the skin, but that he had been unable to find them in time for the meeting. He was, however, of the impression that Dr. Duhring's statement as to the greater frequency of the round-celled form was correct. The general history of these neoplasms is that they are most common after the fortieth year, that they soon ulcerate, and that they exhibit a decided tendency to recur after removal. With regard to metastases and the final termination he was quite in the dark, as he had been unable to follow his cases. He thought that the apparent rarity of the disease was explicable by the fact that tumors usually come under the care of surgeons, and not of dermatologists. He would call Dr. Duhring's attention to Billroth's "Chirurgische Klinik," in the several volumes of which publication about twenty cases of sarcoma of the skin of various portions of the body are detailed.

Dr. Duhring called in question the propriety of the term "sarcoma of the skin," and thought that its primary seat had been the subcutaneous connective tissue.

Dr. Gross said that there could be no doubt that it arose from and was limited to the skin, and stated the reasons for his belief.

Dr. Formad said that he had lately examined one of many nodules situated in the skin, which had been removed and sent to him by Dr. Nash, of Norfolk, Va. The microscope demonstrated that the growths consisted of round-celled sarcomatous tissue, originating in the true skin. *December 9th*, 1880.

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### 16. *Tumor of shoulder.*

Presented by Dr. J. M. Barton, for Dr. L. H. Adler.

The growth which I here present for Dr. L. H. Adler, of this city, was removed by him on the 24th instant from the shoulder of a married lady 28 years of age. It was covered only by skin and superficial fascia, but the deeper portions were imbedded in the trapezius muscle. It had a distinct capsule which was closely adherent to the muscle, and had to be removed by a tedious dissection. It had no bony attachments. The growth was noticed for the first time about two years ago, the patient stating that it was nearly as large then as now. It has never been the seat of pain, and the general health of the patient has been unaffected. The skin over the tumor was perfectly healthy. It was situated on the anterior edge of the trapezius muscle opposite the middle of the clavicle. *June 22d*, 1881.

## IX. SPECIMENS FROM THE LOWER ANIMALS.

### 1. *Hairy papilloma of conjunctiva of an ox.*

Presented by Dr. H. C. Boenning.

This eye was presented to me four weeks ago by a butcher, who regarded it as a great curiosity. While purchasing cattle at the drove-yard, the attention of the gentleman referred to was called to a strange appearance of the right eye of a steer a short distance from the beeves he had selected. Upon closer examination, a hairy tumor was seen protruding through the palpebral fissure. The growth was carefully examined by the cattle-dealers and butchers present, who annually see many thousands of cattle, and all concurred that they had never seen anything of the kind before. The animal was purchased, killed, and the eye brought to me, the gentleman stating that, though the growth was touched with a skewer while the animal was still on the hoof, it did not seem to possess any sensibility, the animal remaining motionless. The hand was also waved to the right of the beast below the level of the eyes without any recognition on the part of the steer; the same motions, when brought within the limits of vision, occasioned turning of the head and an effort to evade the threatened blow. The fresh specimen presented the following appearances. Over the lower, outer portion of the cornea, extending slightly upon the sclerotic, was an ovoid, reddish growth covered with hair of the same color. The growth occupied an area of about a silver dime, was firm in feel, and creaked when cut. It extended from near the centre to a few lines beyond the insertion of the cornea, was about seven lines in diameter, and encircled by a groove of, probably, retracted tissue, this separating a rim of tissue identical with the rest of the morbid structure. A vertical section demonstrated to the naked eye its conjunctival origin. The tumor did not infringe upon the cornea or sclerotic proper. At least one hundred hairs grew from the tumor; some of them were seven lines in length, the remainder varying from three to five lines in length. Section of the fresh specimen showed a moderate amount of epithelium, the rete poorly developed and the

papillæ small, though larger, I think, than shown in the sections kindly made by Dr. Longstreth this morning after about one month's maceration in alcohol. The sub-papillary layers were composed of dense fibrous tissue containing numerous hair-follicles, each containing a non-medullated hair. Numerous sebaceous follicles existed, their ducts emptying both on the surface of the growth and into the follicles. No nerves were visible. This tumor was doubtless congenital. The histogenesis is interesting. The conjunctiva overlying the cornea is without papillæ; hence the impossibility of such a growth containing some of the elements of true skin developing after birth. I might mention that no attachments existed between the tumor and the eyelids. *December* 23*d*, 1880.

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2. *Ulceration of the larynx of the alligator.*

Presented by Dr. C. Seiler.

Dr. C. Seiler said that during his recent stay in Florida he had occasion to examine the vocal apparatus of the alligator. To his surprise, in almost every instance there was more or less ulceration of the epiglottis, etc., in some cases amounting even to the total destruction of this structure. He had discovered that these lesions were due to a small variety of leech abounding in the Florida rivers. In some instances he had detected these creatures *in situ*, and the surfaces left upon their detachment were so exactly similar to those seen in the other supposed specimens of ulceration, that he was compelled to conclude that all were due to the same cause, viz., leech-bites.

*March* 10*th*, 1881.

## X. MISCELLANEOUS.

1. *On some of the causes determining the nature of the effusion in inflammation of the visceral pleura, with remarks upon the prophylactic treatment of empyema.*

Read by Dr. Frederick P. Henry.

In order to an understanding of the proper subject matter of this paper, some preliminary remarks concerning the physiology of respiration and the pulmonary circulation are absolutely necessary.

Among the numerous aids to the circulation of the blood, accessory to the propulsive power exerted by the heart, that afforded by respiration is mentioned by most physiologists. With each inspiration a *vis a fronte* is evoked, without which it would almost seem as if the sluggish current in the venæ cavæ might, in certain states of adynamia, proceed to complete stagnation. No delicate experiments are needed to demonstrate this inspiratory force. When abolished by holding the breath, the veins of the head and neck swell visibly; while, on the other hand, the danger of the entry of air into the veins is one against which all surgeons are on guard during the performance of operations in the cervical region. Although acknowledged as a valuable aid to the general circulation, the influence of this force upon the lesser or pulmonic circulation is not so manifest, and chiefly, perhaps, because the necessity of its assistance is not so apparent, owing to the comparatively short circuit traversed by the blood. Certain well-marked anatomical peculiarities of the pulmonary vessels offer strong presumptive evidence in favor of the view that inspiration is an important aid to the pulmonary circulation. These are the straight course of the vessels and the absence of anastomoses—facts, however, which might with equal propriety be adduced as favoring the view that the heart is alone concerned in the propulsion of the blood, did not experiments definitely decide the question. Ludwig[1] found that blood injected into the pulmonary artery passed with difficulty when the lung was col-

[1] Wagner's Manual of General Pathology, p. 157.

lapsed, quickly when it was moderately distended with air, and when fully distended the stream at first passed quickly but soon became slow. It is thus seen that the condition of lung most favorable to the pulmonary circulation is that of moderate distension, which obtains, in life, during the inspiratory act of tranquil respiration; while the condition least favorable is that of collapse,[1] an approximation towards which is made, during life, at the close of expiration.

Some further remarks concerning the mechanism of respiration cannot be dispensed with.

"The quantity of air which is habitually and almost uniformly changed in each act of breathing is called by Mr. Hutchinson *breathing air*. The quantity over and above this which a man can draw into the lungs in the deepest inspiration he names *complemental air:* its amount is various. . . . After ordinary expiration, such as that which expels the *breathing air*, a certain quantity of air remains in the lungs, which may be expelled by a forcible and deeper expiration; this he terms *reserve air*. But even after the most violent expiratory effort the lungs are not completely emptied: a certain quantity always remains in them over which there is no voluntary control, and which may be called *residual air*."[2]

The above quotation deals with facts which, once presented, are admitted by all, and, for our present purpose, is valuable as affording a classification and nomenclature of the different volumes of air, stationary and fluctuating, concerned in the act of respiration. I prefer the term "tidal air" to that of "breathing air," and will substitute it in this paper.

In the earliest stages of pleurisy the nature of the respiration is altered. The costal type is, as far as possible, changed to abdominal, while the respirations are shallow and, consequently, increased in frequency. This is done instinctively, with a view to mitigate the pain produced by friction upon the inflamed pleura. Now it will be found, on experiment, that there are two principal methods by which the chest-walls can be maintained almost immovable and the respiration be carried on chiefly by the diaphragm.

In the first method the thorax is expanded; the inspiratory muscles are in a state of tonic contraction, and the gaseous interchanges are

[1] By the term "collapse" is here meant the post-mortem condition of a healthy lung removed from the thoracic cavity.

[2] Kirke's Handbook of Physiology.

performed, not by means of the tidal air or normal respiration, but through the medium of what is normally the complemental air, and the reserve air is increased by the addition of the normal tidal. The lungs and the entire pulmonary circulation are permanently maintained in a position closely approximating to that which exists normally only at the height of inspiration.

The second principal method by which diaphragmatic respiration may be carried on while the costal type is suppressed is as follows: the inspiratory muscles are relaxed; the thorax consequently is retracted, and the shallow and rapid respirations are carried on either entirely by the normal reserve air or partly by it and partly by a portion of the normal tidal air. In either case the permanent condition of the lung and of the pulmonary circulation closely resembles that which exists normally at the close of expiration. This method is not so easily imitated as the former. For its proper performance a sharp pain in the side is essential; nevertheless any one who will make the experiment, especially in the recumbent position, will, I think, readily convince himself that such a mode of respiration, even in health, may be maintained for a considerable length of time. Of these two types of diaphragmatic respiration, the latter I believe, both on *a priori* grounds and from observation, to be the one that exists in all cases of acute pleurisy. The former type is rendered impossible by the great fatigue induced by tonic contraction of the inspiratory muscles, and the pain induced by tension of the inflamed pleura.

We have now to consider the state of the pulmonary circulation during expiration, and the probable effect of a continuance of that state in inflammation of the pleura.

Ludwig found, as has already been stated, that blood injected into the pulmonary artery passed with difficulty when the lung was collapsed—that is, removed from the body. The amount of air contained in such a lung varies, other things being equal, according to the rigidity of the tissues composing it, being less in youth than in old age; but the condition of such a lung is not so far removed from that of the living lung at the close of expiration as might at first be supposed, "for, after death, the lungs are not so empty of air as after forced expiration during life."[1] We may, therefore, take this experiment of Ludwig as a guide to our inquiry. In stating that the blood passed with difficulty, no opinion or surmise is expressed as to the seat of

[1] Marshall's Physiology, American ed., p. 819.

obstruction, nor is there any statement, in Wagner's quotation, as to the kind of blood used in the experiment. The obstruction may have been in the arteries, capillaries, or veins. If arterial, it would be followed, in the living lung, by venous congestion, as has been shown by Cohnheim in his experiments upon embolic processes. If capillary, it would be equally followed by venous congestion, although with more participation on the part of the smaller arteries. Obstruction to the flow of blood at any point before the origin of the veins will lead to venous congestion. For proof of this I would refer to what takes place in the disease-process known as cholera kidney. In the kidney, with its double set of capillaries, there is, in these minute vessels, an amount of resistance to be overcome greater, probably, than exists in any other vascular district of the body. It is in the capillaries of the gland that the first obstruction to the low blood-pressure of cholera is opposed, and this is immediately followed by venous hyperæmia. Finally, if the obstruction be venous, it will first affect the distal portion of the veins, and this would occur all the more readily as the pulmonary veins are destitute of valves.

I have thus endeavored to show that the lung assumes an expiratory position when the serous covering is inflamed; that, in this position, there is obstruction to the pulmonary circulation; and, finally, that this obstruction is especially manifested in the primary venous radicles —that is to say, in that portion of the vascular system where our present knowledge teaches us the most essential part of the inflammatory process occurs, the migration of the white blood-cells.[1]

It is not pretended that inflammation is in any sense of the word a mechanical process, but it is firmly held by the writer that there are certain physical properties of the different tissues and organs that influence its progress and termination, if not its origin, and that they are not to be excluded from a proper consideration of its therapeutics. I have elsewhere shown that a high degree of elasticity is possessed by the kidney; and that this property has a regulating influence upon the secretion in health, and is not without influence upon disease-processes, I think highly probable. The elasticity of the kidney was proved by a case in which the gland was the seat of a large inspissated abscess, and in which there was complete occlusion of the ureter. The pus, when fluid, must have occupied a much larger space, nevertheless the remaining cortex of the gland was closely applied to the inspissated

[1] See Green's Pathology, 3d London ed., p. 208.

pus, and its surface was not puckered. Nothing but elasticity could entirely account for such a condition. My concluding remarks upon this subject were as follows: "It must be borne in mind that the ureter was occluded, and, therefore, with the absorption of the exudation there was, as it were, a constant tendency towards the formation of a vacuum, which was prevented by the retraction of the encapsuling kidney. *It has clung to the disappearing exudation as the thoracic walls cling to the retiring lung.* In short, there are three factors capable of producing such a result, namely, the elasticity of the organ, the suction force above described, and, perhaps, some degree of cicatricial contraction."[1] The absence of puckering of the surface went to prove that elasticity was the chief factor concerned.

In considering pleuritic effusions I propose to treat of two varieties only, the purulent and the non-purulent, leaving out of consideration the hemorrhagic form, which is generally symptomatic of the hemorrhagic diathesis, of tubercular pleuritis, or of disease-processes in the pseudo-membrane analogous to those of pachymeningitis hæmorrhagica.

No sharp dividing-line can be drawn between purulent and non-purulent effusions, the difference between them depending solely upon the different quantities of pus-cells which they contain. In a clinical point of view, however, the difference is so great that two distinct affections are treated of, although their primary pathological processes are, in most cases, identical. It is rare that a pleuritic effusion is purulent from the start. Fraentzel says that "primary purulent pleuritis is of extremely rare occurrence. In almost every case the effusion is at first fibrino-serous, and it is during the subsequent course that it becomes, sooner or later, purulent, and this may occur as early as the first week."[2] The form of the disease in which the effusion is from the beginning purulent is known as "pleuritis acutissima," but it is exceedingly rare. Fraentzel had observed only three cases of it, although his attention had been called to the affection by Traube in the year 1860.[3]

Since, then, in the great majority of cases, pleuritic effusions are at first non-purulent, a consideration of the causes leading to the purulent change is evidently of great importance, as through it alone can a treatment designed to prevent such change be rationally instituted.

[1] Path. Soc. Trans., Phila., vol. vi. p. 90.

[2] Ziemssen's Cyclopædia, vol. iv. p. 611.

[3] See foot-note on p. 603, vol. iv., Ziemssen's Cyclopædia.

The earliest manifestation of inflammation of the pleura is hyperæmia of an active nature, accompanied with exudation of liquor sanguinis, emigration of white blood-cells, and increased activity of the endothelial elements. The inflammatory stasis that speedily supervenes in the capillaries causes, by its obstruction, as I have endeavored to show in the foregoing pages, and as is proved by experiments upon the transparent tissues of animals, an early engorgement of the primary venous radicles, and this engorgement is still further added to by the expiratory position in which the lung is maintained on account of the pain. The conditions, therefore, are in the highest degree favorable to the emigration of white blood-cells,—that is, to the production of pus. The fact that the pulmonary vein contains arterial blood may be a provision of nature against the tendency to cell-migration, but it is the fibrin now flowing upon the pleural surface that opposes the principal barrier. Whether fibrin be a simple or compound substance; whether its origin be in the blood, as held by Rindfleisch, or in the inflamed tissues, as held by Virchow;[1] whether it be nutritious or excrementitious, it is certain that this substance is apt to preponderate in the inflammatory products of serous inflammations occurring in robust persons. The migrating cells are entangled in its meshes before reaching the pleural cavity, and one of its offices here, as in the case of incised wounds, is undoubtedly the formation of a "*scaffolding*," by means of which the building up of the cells into fibres and bloodvessels may take place.

A further proof that fibrin interposes a barrier to suppuration is found in the fact that the first step in the conversion of a fibrino-serous into a purulent effusion is the dissolution of the fibrin deposited upon the pleura. In discussing the earliest steps of this conversion, Rindfleisch remarks, "Large shreds of the pseudo-membrane are loosened and detached from their resting-place, then to liquefy into pus."[2] Without pausing to criticize the expression "liquefy into pus," which may possibly be an incorrect rendering of the original text, I would particularly call attention to this first stage of empyema thus described by this eminent authority in pathology, as allusion will again be made to it in discussing the prophylactic treatment of empyema.

During the stage of the disease above referred to, the lung, owing to the pain, has been maintained in an expiratory position, and the

[1] Rindfleisch's Manual of Pathological Histology, p. 259, Philada. edition.
[2] Path. Hist., Am. ed., p. 269.

rapid and shallow respirations have been carried on chiefly by the diaphragm. This is best observed in women, in whom the normal type is "superior costal." At the same time the rhythm of respiration is altered. According to Burdon-Sanderson,[1] the rhythm of normal respiration is as follows: the whole respiration period being 15, inspiration occupies 4, expiration 2, and the pause 9 parts. In the jerky respiration of the acute stage of pleurisy it needs no other instruments than hand, eye, and ear to perceive that this pause is both absolutely and relatively shortened, and that inspiration and expiration are of about equal duration. In so far as the pause at the close of expiration is shortened, the effect in pleurisy upon the circulation, and through it upon the effusion, is conservative; nevertheless this is more than counterbalanced by the permanent expiratory position assumed by the lung.

There is another cause of the expiratory position of the lung, whose influence, at first scarcely felt, gradually increases until its limit, varying in different cases, is reached. This is the effusion, which also for the most part proceeds from the veins. According to the views advocated in this paper, the mechanical, compressing action of the effusion may be instrumental in propagating the inflammation. For example, we will suppose a pleurisy beginning at the apex of the lung attended with a moderate effusion. This effusion gravitates to the bottom of the pleural cavity, already predisposed to inflammation by the congestion produced by the expiratory position of the lung assumed on account of the pain. The effusion, by exerting a greater compression, will increase the congestion, and, in combination with other causes, may actually excite inflammation. If this theory be correct, a secondary pleurisy would arise at the base of the lung, and, proceeding from below upward, unite with the primary disease proceeding in the opposite direction. Such a theory, if true, would be hardly susceptible of proof, because it would be impossible to exclude the irritating qualities of the effusion.

The fact that empyema is generally, at the beginning, a pleurisy with fibrino-serous effusion, is suggestive in more ways than one. I have alluded to one of the methods by which this change may take place,—a change, I repeat, which, in a pathological sense, is merely one of degree. This is by the breaking up of the barrier of fibrin. Any cause leading to this unfortunate result will tend to convert the

[1] Marshall's Physiology, Am. ed., p. 814.

disease into empyema. These causes are such as interfere with the healing of wounds by first intention, where also a certain amount of fibrin is necessary to support the cells during their metamorphoses. Among these causes may be mentioned pyæmia, septicæmia, and the infectious diseases. If with any such, or similar conditions of dyscrasia, there is also an increase of the white blood-cells, the change to empyema is still further favored. Such a condition is found in scarlatina,[1] and in this affection empyema is by no means rare. I have met with a case, fully reported elsewhere, in which empyema supervened upon a prolonged attack of intermittent fever,—that is to say, during a condition of the system in which it is almost absolutely certain that there was not only increase of the white corpuscles but also diminution of the red.

Virginia S., æt. 24, was admitted to the Episcopal Hospital on September 27, 1874. Her illness, which had lasted about a year, began with a chill, followed by pain in the left side, cough, and swelling of feet and legs. She had suffered from *chills and fever* during the entire summer of 1873. It is, of course, necessary to be careful not to confound such *chills and fever* with the similar symptoms that often attend the process of suppuration. There was no doubt, in the case referred to, that the intermittent fever was of malarial origin, and the state of the blood thereby produced was manifested not only by the effusion of pus into the inflamed pleura, but also by the occurrence of œdema of the lower extremities at a very early stage of the case. The chills and fever were not the hectic of phthisis, for at the post-mortem examination of the case, which was made January 7, 1875, the only indications were "a few caseous nodules at the right apex"—an amount of disease often found at the autopsies of subjects that had not been considered phthisical during life. The empyema was on the left side, and the "lung was compressed into a firm oblong mass, not much larger than the fist, and lay in the upper and back part of the cavity, being closely bound down to the vertebræ.[2] This lung was carnified, but not the seat of caseous or tubercular deposit. Indeed, the occurrence of large effusion—and at the third tapping of this case, for the purpose of inserting a drainage-tube, five quarts of pus were removed —the occurrence, I repeat, of large effusion argues in favor of a comparatively healthy condition of lung at the beginning of the pleurisy,

[1] Ziemssen's Cyclopædia, vol. ii. p. 234.

[2] Path. Soc. Trans., Phila., vol. v.

for it is self-evident that a solidified lung forbids any great accumulation of fluid in the pleura. A certain amount of inflammation of the lung exercises a conservative influence against the occurrence of effusion, and it is the opinion of the writer, founded upon the clinical study of pleurisy, that in acute cases, where the fibrinous element largely preponderates and the fluid effusion is small, there is generally a decided amount of inflammation of the peripheral parenchyma of the lung; that is to say, the case is one of pleuro-pneumonia. When there is large effusion, on the other hand, the inflammation is more strictly confined to the pleura. I admit, however, that this interesting point is one demanding further study.

But the most interesting question in connection with the origin of empyema is, whether a serous effusion may, by its mere pressure, keep up such a degree of venous congestion as to lead to sufficient cell-emigration to convert the effusion into pus. That a certain degree of pressure has the effect of keeping up the inflammatory process until the subserous connective tissue is extensively involved, and a granulating, pus-discharging surface formed in the site of the pleura, is, I think, highly probable, but the degree of this pressure cannot be ascertained even for a single individual, and it probably differs in all. Pressure of the effusion, independent of the continuance of inflammation, according to its amount, produces one of two effects. One degree, by the peripheral venous congestion it produces, promotes effusion; a still greater degree prevents it entirely, and the fluid arrives at a state of stagnation, by which I mean that it is neither added to nor diminished. This is the condition met with in many chronic pleurisies when they first come under our notice. The disease-process appears to have come to a complete stand-still, and, if we choose to so consider it, this counter-pressure upon the bloodvessels, by which further effusion is prevented, is an instance of the much-vaunted "*vis medicatrix naturæ.*" Apart from its absorption, the duration of this stationary condition of the effusion depends upon two things: the power of resistance of the patient, and, if the effusion be purulent, the supervention, or not, of necrosis of neighboring tissues. If such necrosis is accompanied with inflammation, the fluid may increase before it perforates; in the absence of fresh inflammation, there is no further effusion. Withdrawal of the fluid, or a portion of it, by relieving the walls of the vessels from counter-pressure may again place them in a condition favorable to further effusion. On the other hand, it may also place them in a condition favorable to absorption; and that

this condition is probably largely mechanical, is proved by the following quotation from Dr. Bowditch's paper on Pleuritic Effusions:[1] "Dr. Wyman assures me that he operated in one case of chronic pleurisy eight or ten times, and there was no permanent relief until the last puncture, at which less fluid was drawn off than at any previous one, and yet the patient apparently obtained greater relief from it than from all the others."

Dr. Bowditch attributes the recurrence of effusion after repeated tapping to a "habit of effusion gained by the pleura." From this "habit" he infers the early removal of the fluid, and is, I think, in most cases, entirely correct in his inference. Still, the argument founded on "habit," although leading to a correct result, is not sufficiently convincing: one naturally demands something more in the way of a reason. That the degree of pressure upon the exterior of the walls of the vessels has a great influence upon the process of absorption, either inhibiting it, not opposing it, or favoring it—as it is well known that pressure favors osmosis—is, I think, indubitable. At the same time, this opinion, it must be remembered, is founded upon the supposition that the walls of the bloodvessels are healthy; and that they are so, after prolonged compression of the tissue surrounding them, is highly problematical. Although not strictly analogous, I may here refer to Cohnheim's experiments in interrupting the circulation through the renal artery. After removal of the ligature the veins became dilated, and from them there issued an abundant emigration of white blood-corpuscles together with blood-plasma.[2] It was an alteration in the nutrition of the vessels that led to this result, and a similar alteration may attack the walls of the bloodvessels of the lung after prolonged interference with their supply of blood by a large pleuritic effusion. Such a disease of the bloodvessel-walls would serve to explain the numerous cases in which what was a serous effusion at a first tapping or aspiration has been found to be purulent at a second or third. These changes in the walls of the veins, consequent upon a prolonged interference with the circulation, are of an extremely delicate nature, for Cohnheim "was never able to discover the slightest alteration in the minute histological structure of vessels that had been thus a long while deprived of the contact of blood that should have circulated through them."[3] The fact of the conversion of a serous

[1] Am. Journ. Med. Sci., April, 1852.
[2] Ziemssen's Cyclopædia, vol. xv. p. 230.
[3] Ibid., p. 224.

into a purulent effusion after operative interference is notorious, and has been attributed to the admission of disease-germs, and to the traumatic irritation of the pleura, but neither of these explanations can withstand criticism, for similar interference with other serous cavities is not followed by the same result. The effect is partly mechanical, the counter-pressure being withdrawn to such an extent as to favor exudation, and partly vital, owing to a change in the walls of the vessels, more especially of the smaller veins.

It would appear, on superficial consideration, that if the prolonged duration of the effusion favor its purulent metamorphosis, all cases of pleurisy, after a certain length of time, should be converted into empyemas, or at least that cases of empyema should be of a more chronic nature than those in which the effusion is fibrino-serous. But the question is not so simple. A serous effusion, if sufficiently large to prevent by its counter-pressure any further effusion, may remain serous indefinitely if not interfered with. This is not the case with a purulent effusion, whose tendency is to seek an outlet in the direction of least resistance. I began the analysis of a series of cases with the object of comparing the chronicity of the two affections, but abandoned it after becoming convinced of the negative nature of the evidence thereby afforded. I submit the figures, however, as they possess a certain degree of interest.

In vol. xxvii. of the *Medico-Chirurgical Transactions* is an article on paracentesis thoracis, by Hamilton Roe, M.D., of London, which contains two sets of tables, the first containing thirty-nine cases, being all that had been "published in the English language between the years 1812 and 1832, inclusive," the second containing twenty-four cases of paracentesis performed under his own observation or that of his friends. Of these sixty-three cases twenty-nine were cases of empyema and twenty-six cases of "inflammatory hydrothorax"—that is, pleurisy. Leaving out one case of empyema of seven years' duration and one of pleurisy of three years', and one of sero-sanguinolent effusion, I obtained for analysis twenty-five cases of empyema and twelve of pleurisy, in which the duration of the disease previous to operation was given. The result was that the average duration of the empyema cases was ninety-eight days and that of the pleurisy cases ninety-seven days.

In *Guy's Hospital Reports* for 1877 is an article on empyema, by Dr. James Goodhart, containing a table of seventy-seven cases of empyema treated by operation. It is made up in nearly equal proportions

of cases treated at Guy's Hospital during the preceding ten years, and of similar cases treated at the Evelina Hospital for children since its foundation in 1869. Although not available for any present purpose of comparison, it is interesting as showing the more acute course and greater frequency of the disease in children. Leaving out the cases in which there were sinuses on admission and those in which the duration of the disease was doubtful, I obtained thirty-three cases occurring in children ten years of age or under, and twenty-six in individuals over that age. The first set gave an average duration of forty-nine days; the second, of sixty-nine days.

I am aware that there are numerous sources of fallacy in all such statistics, and it was this conviction that induced me to discontinue my researches in this direction.

It is the opinion of most authorities that the chronicity of a pleurisy is no proof of its purulent nature, or, at least, that it affords no guide to diagnosis. In the volume of the *American Journal of the Medical Sciences* for 1852 are two admirable articles upon the subject of pleuritic effusions, one in the January number, by the late Dr. Pepper, of Philadelphia, the other in the April number, by Dr. Bowditch, of Boston. In discussing their diagnosis, Dr. Pepper says that the chronicity of the effusion affords no guide, while Dr. Bowditch remarks, in the same connection, "The chronic nature of the complaint affords no criterion by which to judge." This is true in a certain sense, not so in another, for in an acute case there is, as a general rule, little question of pus, while in a chronic case this question always arises. The acute cases, with moderate effusion, are generally cured by medicinal treatment. The prognosis of completely uncomplicated pleurisy is good. In fact, there are two well-marked diseases, clinically speaking, under the head of pleurisy, an acute and a chronic affection, and under the second head comes empyema, so that the duration of the affection does afford some guide to diagnosis. That Dr. Pepper believed in the power of time, in combination with other causes, to convert a pleurisy into empyema, appears from the following remark concerning the first case reported in his paper: "In the above case it would appear that the acute pleurisy, contracted in the spring of 1842, was, from neglect or inefficient treatment, allowed to degenerate into chronic empyema." Time may, in most cases, be said to predispose to empyema, and especially in the class of hospital patients, in whom other depressing causes are almost invariably associated. There is one theory regarding the conversion of a serous into a purulent effusion for which time is essen-

tial, viz., that which attributes it, in great measure, to continued subdivision of its contained leucocytes. This theory is held by Fraentzel, and partially admitted by Rindfleisch; nevertheless I am not willing to ascribe to such a cause any great degree of influence in the conversion of serum into pus. If such a cause were commonly operative, empyema would be much more commonly met with. The process of cell-division has its limit, and once the cells are suspended in the effusion, and freed from any further nutritive influence other than that supplied by the fluid surrounding them, that limit is probably very soon reached.

Another cause leading to large effusions of serum, and predisposing to empyema, is probably congenital. The extent of the respiratory movement differs in every individual, and is known as the respiratory or vital capacity; and the point of thoracic equilibrium, the pivot, as it were, upon which these movements turn, of necessity also differs. It is highly probable, for example, that if this centre of movement be congenitally high, and, at the same time, the respiratory capacity be small,—if, in short, owing to the individual idiosyncrasy, the thorax be maintained in a position of inspiratory expansion,—there is a strong predisposition to the development of emphysema. This is the theory of emphysema advocated by Freund,[1] who attributes the inspiratory position to a nutritive lesion of the costal cartilages, although he considers it as acquired rather than congenital. If, on the other hand, the point of thoracic equilibrium be congenitally low, and the respiratory capacity small, a tendency to venous congestion in the pulmonary venous radicles is part of the constitutional inheritance of the individual, and a pleurisy attacking such a subject would, in accordance with the views herewith submitted, be probably attended with large serous or purulent effusion. Corroboration of this theory would not be found in an antagonism between emphysema and pleurisy, if such could be shown to exist, as emphysema also predisposes to venous congestion, for it will be remembered that in Ludwig's experiment, when the lung was greatly distended with air, the blood-stream at first passed rapidly but soon became slow. These two conditions of lung, the inspiratory and the expiratory, in their effect upon the circulation, afford an instance of the meeting of extremes.

It remains to be considered what therapeutical deductions are to be drawn from the preceding general principles. In the present paper,

[1] Ziemssen, vol. v. p. 359.

this question is limited to the inquiry whether we have any means at our command by which we can prevent the degeneration of a fibrino-serous into a purulent effusion.

As regards a prophylactic treatment based upon the shape of the thorax and the want of development of the respiratory muscles, there is little to be said, for if general considerations of health do not lead to the proper development of this part of the body by judicious exercise, the possibility that an attack of pleurisy occurring in such an individual may degenerate into an empyema will certainly not do so.

The earliest symptom of acute pleurisy, pain, is to be combated with a full dose of some preparation of opium. There has been much discussion regarding the influence of pain in disease, the result of which has been, in many cases, the decision that pain, *per se*, exerts a positively injurious effect upon the local affection. Such statements should be received with caution. When, after the administration of a full narcotic dose, an inflammatory disease is speedily recovered from, the first question should be, What is the natural duration of this disease? A few cases of painful inflammation speedily recovered from under full doses of narcotics are not sufficient to settle this important question. There is at least one important principle concerned, which is this: when pain *through its interference with function leads to congestion*, its removal exerts a direct curative influence upon disease; otherwise its removal can exert but an indirect curative influence through the general nervous system. In pleurisy, as I have endeavored to show, pain, by its interference with function, promotes congestion, and its removal, in that it permits of a freer movement of the lung, is the first indication. Such a doctrine would, at first sight, seem opposed to the usual treatment of inflammation by rest, but this rest is always qualified by the phrase, "in the proper position,"—that is to say, in a position calculated to diminish venous congestion. Position is more important than rest.

Experience has already shown the value of opium in the earliest stage of pleurisy, but the therapeutics that is founded upon experience solely, although perhaps the best, does not command universal assent, because the nature of the experience, its amount and quality, have always to be taken into consideration; but if this experience can be shown to have been founded, although perhaps unconsciously, upon the most fundamental pathological processes of the affection, the authority of experience is such as to command assent. Until this is done, valuable time will be lost, and lives perhaps endangered, by mere ten-

tative experiments in other therapeutic directions, by those ambitious to discover specifics, and also by those—a numerous class—who on all subjects instinctively range themselves upon the side of the opposition.

The question of bleeding is one of great importance. That bleeding is beneficial in the earliest stages of pleurisy, in many cases, there can, I think, be no manner of doubt. Such is the universal testimony of most clinical teachers during the first half of this century, and this testimony it is impossible to refute without a resort to the extreme position of a change in the type of disease. But in order that good may result from bleeding, it is not to be resorted to without great discrimination. In the first place, its use should be confined to the first stage. For the purpose of promoting absorption of the effused fluid, we have agents quite as efficacious as bleeding, and free from its disadvantages. The influence of bleeding for good or evil in pleurisy can only be thoroughly appreciated by a close study of the pathology of the disease. In the earliest stage of pleurisy the fibrinous barrier to suppuration is forming upon the pleura, but forming slowly, as it is the nature of fibrin contained in inflammatory blood to coagulate slowly, although the coagulum once formed is of the densest kind, in order to oppose suppuration to the utmost extent. Bleeding will hasten the coagulation of the fibrin, as it is well known that in venesection the blood first withdrawn coagulates more slowly than that last drawn, but the clot will not be so firm. It becomes a question, therefore, whether more will be gained by venesection and the consequent rapid formation of a layer of fibrin of loose texture, than by the slower formation of a denser layer, such as would be the effect of leaving the process to nature. It is a question requiring careful and prompt decision, as after the first twenty-four hours little can be expected from bleeding. Bleeding acts favorably also by diminishing pain and thereby favoring the freer movement of the lung. As regards the form of bleeding, I give the preference to wet cups applied over or near the seat of pain, as in this way probably the greatest relief will be obtained. Bleeding should not be resorted to except in the case of robust individuals. In anæmic individuals, where bleeding cannot be resorted to, the chief reliance is to be placed upon opium and quinia. It has been shown that quinia diminishes the amœboid movement of the white blood-cells, and so it is inferred that it is opposed to suppuration. Whether this be so or not, it is perhaps our most valuable antipyretic and tonic, although its value as an antipyretic in the fever of inflammatory diseases has not been, in my experience, so great as

in the fever of the infectious diseases. I do not think it necessary to administer it in the earliest stage of acute pleurisy occurring in robust people, relying chiefly in those cases upon opium and wet cups.

The prophylactic treatment of the effusion—that is, the treatment designed to prevent its purulent degeneration—is medicinal and surgical. The chief drug to be relied upon is quinia, both with the object of preventing further cell-migration and for the purpose of preventing further activity in the cells contained in the effusion. Surgical treatment is not to be instituted while the effusion is in the acute stage and rapidly progressing. In Bowditch's eighth case,[1] tapping was resorted to on the seventh day of the disease, and thirteen ounces of serous fluid were removed: five days afterwards "the signs of effusion were fully as great as at first." The case recovered eventually without subsequent tapping, was well on the thirty-ninth day, and probably would have done as well under medicinal treatment alone. But when the effusion has ceased and has remained stationary for some days—it is, of course, impossible to lay down any absolute rule as regards time—the removal of a small proportion of the fluid by aspiration is indicated. My own experience would lead me, in the absence of any alarming symptoms of dyspnœa, to wait at least a week after the effusion has become stationary, before resorting to aspiration. The energetic employment of counter-irritants, diaphoretics, such as jaborandi, or its alkaloid used hypodermically, and diuretics, is generally followed by a rapid reduction of the serous effusion, and a gradual restoration to health, provided the case has been properly treated from the beginning.

The treatment of purulent effusions does not come within the scope of this paper.

Dr. Tyson said one well-known cause of pleuritic effusion had not yet been referred to by Dr. Henry—that is, a change in the composition of the blood, such as occurs in Bright's disease, where there has been a loss of albumen, and a consequent watery state results. Such blood is very prone to pass out through the vessels into serous cavities, as well as into the subcutaneous connective tissue.

Dr. O'Hara thought that the theory proposed was too mechanical to explain all the cases of pleural effusion. He had seen cases of empyema discoverable while the patient was walking about, without any previous notable symptoms of pain, fixity of the breathing apparatus, etc. He had seen, two years ago, notably one case of pleural effusion

[1] Am. Jour. Med. Sci., April, 1852.

without apparently any other symptom in the patient than general debility and a gradual filling up of the left pleural sac. It became distended to a degree requiring removal, which, with the assistance of Dr. Willard, was done by the aspirator to the amount of five pints. In three weeks this, with tonics, enabled the patient to go to his work. About four weeks afterwards the same thing occurred on the right side, and three pints of fibrino-serous fluid were removed. The patient has remained well since. In this case there was no renal, cardiac, or liver trouble. He had slight cough, and from his debilitated condition tubercle was feared, but after-events showed this not to be the case. This case caused him to think that the portion of the nervous system presiding over secretion might, by a kind of paresis, cause an alteration in the quantity or quality of the ordinary fluid of the pleural sac. He had an idea that something might take place similar to that which you have from nerve-irritation, as in the exudation of herpes zoster.

Dr. Mills said, in regard to the influence of the nervous system determining effusions, that it was known that in disseminate sclerosis, involving both brain and spinal cord, the patients have apoplectic attacks in which the lung symptoms become prominent; he had seen attacks like pneumonia in the first or beginning of the second stage, or like acute pleuritis, post-mortem examination revealing large effusions.

Dr. Wilson related a case illustrating the length of time that a pleural effusion may persist without becoming actually purulent. It was that of a young man who was taken ill with all the symptoms of an acute pleurisy. Shortly afterwards the signs of a large effusion of the left chest supervened. He was under treatment without operation, and in very poor health, for more than two years. At the end of this time, namely, in April, 1877, he came under Dr. W.'s care, and the fluid was evacuated by aspiration. It presented to the eye the appearance of a slightly-turbid serum, and under the microscope plates of cholesterin and leucocytes. The quantity was a trifle more than five pints. Eight months later, it had reaccumulated, and was again removed in the same manner, to the amount of a quart. The fluid at the second aspiration was of the same character as at the first—sero-purulent. Since the second tapping there has been no reaccumulation.

It would be impossible to discuss fairly Dr. Henry's ingenious and elaborate paper at present, as it presented many points that were novel and not to be fully appreciated except after careful consideration. He thought that those who were accustomed to study pleural effusions from the clinical stand-point looked upon the character of the effusion as, to

some extent at least, determined by the condition of the blood, or, at all events, by something in the nutrition of the individual. It was his impression that conditions of cachexia, or even diathetic states, influenced unfavorably the character of the product in serous inflammation; and he narrated two cases of pleurisy in which an early operation led to the evacuation of fetid pus, both of which occurred in persons belonging to highly consumptive families.

Dr. Seiler said he had recently made a post-mortem examination in which there was found in the right pleural cavity a large amount of creamy pus; the lung was collapsed, lying against the vertebræ. The patient had died of diarrhœa. No symptoms of lung-trouble had been detected during life, with the exception of a change in the voice. This he thought an evidence of a large amount of fluid existing in the pleural cavity for some time without giving rise to any symptoms.

Dr. Eskridge said, that the general circulation is influenced by inspiration and expiration, there can be no doubt; but that the pulmonic or lesser circulation is influenced by either, exclusive of the chemical change effected by these phenomena, he doubted.

The rapidity of the general venous circulation is increased during inspiration, because there is an effort to establish a vacuum in the thorax, towards which the venous blood is flowing. The rapidity of the general arterial circulation at the same time is decreased (or, what is the same thing, the pressure in the aorta is lessened), because these vessels carry the blood from the cavity of the thorax, in which, by the inspiratory effort, a vacuum is attempted.

As expiration upon the general circulation has just the opposite effect to inspiration, the rapidity of the general venous circulation is decreased and the arterial increased during expiration.

Since the general circulation is mainly outside of the cavity of the chest, and has its rapidity affected by respiration on account of certain mechanical phenomena which take place during this process, as the blood either flows to or from this cavity, he could not see how the mechanical movements of respiration could affect a circulation like the pulmonic, which is wholly within the chest.

We know that the blood well supplied with oxygen will circulate more freely than blood loaded with carbonic acid gas. In the healthy adult, from eighteen to twenty cubic inches of air enter and leave the lungs during every respiratory act. During the pain of pleurisy the chest-walls are not allowed to expand so freely as in health; less air, therefore, enters and leaves the chest during respiration, the tissues

soon call for oxygen, the blood is loaded with carbonic acid gas and effete matter, the number of respirations to make up for the deficiency of oxygen is increased; but, as each inspiration is now necessarily short, the blood is not sufficiently oxygenated or relieved of carbonic acid gas to prevent the latter from increasing the morbid action already going on in the congested and inflamed pleura.

Dr. E. did not believe that the mechanical movements of respiration directly affect the rapidity of the pulmonic circulation.

Dr. Nancrede said in regard to duration being a cause of the fluid becoming purulent, he had had charge of a case in which there had been severe traumatic injury of the chest-wall, resulting in considerable general emphysema and collapse of the lung. The symptoms of the consequent pleuritis had been very violent. The fluid became encysted, and remained stationary for four months. In consultation with Dr. Starr, it was determined to draw off a small amount of the fluid with a hypodermic syringe for diagnostic purposes, when the exudation was found to be clear and limpid. No further operation was done, and the man recovered.

Dr. Formad suggested the examination of pleuritic effusions for albumen, believing that the estimation of the quantity of the latter would determine the prognosis of the serous effusions. He had several times examined hydrocephalic effusions tapped by Dr. Detmold, of New York, without finding any trace of albumen; and also, in a similar case operated upon by Dr. Pepper, at the University Hospital, he found only a trace of albumen. Having also a number of fluids tapped from cases of pleuritic effusions, ascites, hydroceles, etc., he ascertained that, while some of them were loaded with albumen, in most of the fluids there was only a small amount, and in a few no trace even of albumen could be detected. Two cases of pleuritic effusion, in which a large quantity of albumen was present, he knew to have terminated fatally. It is a known fact that the quantity of albumen in exudations is always in direct proportion to the intensity of the inflammation, and that exudations (sometimes in large amount) in congestive conditions contain little or no albumen, albumen being dependent upon the number of migrated cellular elements, which are few in the latter condition. The albumen in cystic fluids is dependent upon another source, viz., that of a mucoid degeneration of tissues.

Dr. Tyson thought Dr. Formad's statement in the main correct, but it went too far. That albumen is invariably present in inflammatory effusions is undoubtedly correct, and that it is absent in many non-

inflammatory effusions is also correct; but non-inflammatory effusions also occur which contain a considerable proportion of albumen. He was aware that the view of Dr. Formad was to be found in some of the text-books, but thus expressed it is erroneous.

Dr. Hodge called attention to the fact that in ovarian dropsy we have a fluid in which the amount of albumen is very abundant without there having been any symptoms of inflammation of the cyst.

Dr. Henry remarked, in conclusion, that his paper had reference solely to inflammatory effusions. It was evident that there was some misunderstanding in the discussion, although those who had heard the whole paper would probably recall the fact that nothing whatever had been said in regard to non-inflammatory effusions. Dr. Henry now regretted that, for the sake of brevity, he had caused only a portion of the title of his paper to be printed on the notices of the meeting. It was announced as a paper on "Some of the Causes Determining the Nature of Pleuritic Effusions," which would, of course, include hydrothorax.

The discussion had wandered very wide of the point. The proper method of refuting the views advanced was to show that the experiments of Ludwig upon the pulmonary circulation and those of Cohnheim upon embolic processes are unreliable, or to point out some flaw in the arguments, which are entirely based upon these experiments. This had not even been attempted. Exceptional cases of pleurisy and empyema, attended with remarkably few symptoms, cases which are now readily recalled to the memory on account of their exceptional nature, are brought forward by way of argument, but are hardly entitled to be so received.

In reply to both Dr. O'Hara and Dr. Wilson, Dr. Henry would say that, while giving prominence to one set of causes determining the nature of pleuritic effusions, he had by no means omitted to refer to constitutional conditions as concerned in the conversion of a sero-fibrinous into a purulent effusion. "Pyæmia, septicæmia, and the infectious diseases" are distinctly mentioned as predisposing to such a change, and it will surely be recalled by some one present, that one case illustrating the effect of constitutional causes upon the nature of the effusion is referred to at some length. Finally, in reply to the remarks of Dr. Eskridge, who so emphatically dissents from the results of Ludwig's experiments, Dr. Henry would say that a mere statement of unbelief in the experiments of so distinguished a physiologist is not sufficient to refute them. *September 25th*, 1879.

## 2. *Some points in the pathology of tubercle.*

Read by Dr. J. M. Da Costa.

In attempting to put together some thoughts on the pathology of tubercle, it will be necessary, however briefly, to refer to the unsettled state of the question in the best medical minds of the day. Immediately following Laennec, nothing could have appeared more firmly fixed than the doctrine he so clearly enunciated. It was impossible to doubt that tuberculosis was a specific disease. To have misgivings as to the nature of consumption and its constant association with tubercular destruction was to appear to return to barbaric darkness. Not to separate with clearness the different forms of tubercle was to forfeit all claim to be a pathologist. But we all know what has recently happened. The German iconoclast has been at work. Nobody likes to speak now of tubercular diathesis, of tubercle being a constitutional affection. It is for the most part simply the result of a local inflammation; and cheesy matter, infective process from absorption, irritation in structures abounding in lymphatic tissues, are the complacent phrases of the day, which satisfy most as much now as diathesis, constitutional condition, specific deposit, satisfied most not many years since.

And the local view, if such it may be called, once adopted, has brought with it scores of interesting observations on the inoculation of tubercle; its artificial causation; its production in the lung by inhalation of both tubercular and non-tubercular substances—observations which are warmly discussed, criticized, adopted, rejected, explained, explained away, and the uncertainties connected with which, quite apart from the other difficulties of the subject, are the cause mainly of the generally disturbed condition of the whole inquiry.

Underlying these observations, or at least closely connected with them, lies the vital question, What relation does tubercle bear to the inflammation? And it is this question particularly that I desire to examine with you a little more fully to-night, and concerning which I shall venture to offer the result of some researches and reflections.

As a necessary introduction, I shall have to examine the evidence on which we pronounce a mass to be tubercular; in other words, what its minute structure as shown by the microscope is. And, to avoid any confusion at the threshold of our inquiry, let me speak of that which

we find in undoubted tubercle, in the little, hard, miliary bodies, which may afterwards become aggregated into larger, gray masses. In them we encounter three elements :—

*Medium-sized, rather shrivelled cells*, not very regular in outline, consisting of finely-granular, dense material, with a nucleus small in proportion to the cells, or with several nuclei of similar character. They were once regarded as significant of tubercle, but they are now supposed to be swollen epithelial cells which have undergone retrograde metamorphosis. Mixed up with them are cells less dense and like ordinary epithelium, small cells, and a great deal of granular material of doubtful origin.

*Giant cells.* These consist of large, many-nucleated cells, which are found at rather an advanced stage of tuberculosis, and are very marked in the acute form. They are of spheroidal shape, and somewhat irregular outline. Great stress has been laid on them as significant of tubercle, but they have been met with in deposits in various tissues of the body, in scrofula, in syphilis, and in merely hyperplasic lymph-glands of those perfectly free from tubercle.[1] As they grow they send out long, branched processes. With Klein, I believe them to be excessively developed or fused epithelial cells.

The structure in which all these cellular elements are found, especially, perhaps, those last described, is *a fine network* like the fine trabecular network in the interior of lymphatic glands; and this led to the belief entertained until recently by Rindfleisch, that tubercle is a lymphoid growth. But this is not stating the whole of the manner and arrangement of the cells in tubercle. They are found in the lungs filling the alveoli and infiltrating—generally as small, round cells—the alveolar walls, and leading to very considerable thickening of the latter.

To sum up, cell-growths by themselves, not peculiar, but representing different grades of development—some still rapidly growing, others shrivelling and full of dense matter; all capable of being washed out of a fine reticulum, or accumulating in masses both within and in the walls of air-vesicles—this structure, this grouping, may be regarded as tubercle. Then there are certain secondary alterations that take place in the tubercular formation and the invaded tissue which must also be mentioned, and which bespeak a retrograde change and low vitality. The main of these changes is a degeneration of the cell-growths, an accumulation of granules and fatty material, and an occlusion of the

[1] Weiss, Virchow's Archiv, lxviii.

pulmonary capillaries, probably from pressure, and here and there a fibroid transformation of the giant cells.

Now, what causes all this? Some still maintain a specific non-inflammatory deposit; some say an inflammatory process of slight intensity; others a specific inflammation. I pick up a recent journal, and see that malaria is at the bottom of all this strange cell-growth and rapid decay. I turn to one of this month, and I find in the front of the periodical an article proving that tubercle has its origin in disorders in the trophic centres, and in the middle pages another, showing that it is an accident, the result of the capillary interference, due to an altered condition of the blood from the presence of yeast. It is almost needless to say that the bacteria are made to explain the peculiar formations, for how could these patient little beings that are bearing so quietly being made the scapegoats of the pathologists of the last half of the nineteenth century escape having charged to them this additional sin? I turn with eagerness to discussions of the subject replete with learning in societies similar to ours, and there is little but negation. It is not this, and not that, say men who are known wherever medicine is cultivated; and you begin to doubt if there is such a thing as tubercle at all, until the first clinic-room you go into—and see the familiar face, hear the cough, and recognize the well-known signs—confronts you with the stern reality of the awful disease. With all these doubts and gropings after the truth, I may be pardoned if I hold fast to the belief that the process, whatever it be, is something special, though something of which we do not hold the key.

To return from this digression to one part of the subject around which much of what is positive in our knowledge has clustered, and which is of most obvious applicability—the relation of these mysterious tubercular formations to inflammation.

Now, we all know how the relation of tubercle to inflammation has engaged the attention of the present generation of pathologists. Yet the consideration of the question long antedated them; and the much-neglected observations of that sagacious thinker, Addison, are really the key-note to many of the views now brought forward under other names. But this is an historical issue, with which we cannot further concern ourselves here. The active discussion of the matter started with the observation of Virchow, that the caseous matter previously regarded as infiltrated tubercle might originate from the fatty degeneration of diverse morbid products, and was non-tubercular; indeed, that the gray granulations alone were tubercular and non-inflammatory.

Niemeyer expanded this thought, and engrafting on it the doctrine of Buhl, of the infection of tubercle, promulgated the view that the lung consolidation and destruction were most commonly inflammatory—the result of the caseous pneumonia—and the tubercle, when met with, quite secondary and accidental. Indeed, to carry out Niemeyer's ideas logically, the inflammation is, in the vast majority of instances, everything, the tubercle nothing.

The doctrine of Buhl has been alluded to, that tuberculosis—as seen, for instance, in its most typical form of miliary tubercle—is due to infection from masses of cheesy material. The infection happens chiefly through the lymphatics. This infection theory led to numerous experiments on animals, with the result that inoculation in rabbits and guinea-pigs with fresh miliary tubercle, with cheesy matter, with the sputa from tubercular patients, has been followed by acute miliary tuberculosis. Moreover, going still further, Cohnheim and Fraenkel have shown that it is unnecessary to inoculate with these special matters, for in rabbits and guinea-pigs the formation of any focus of suppurative inflammation may fill the viscera with tubercles. With reference to these experiments, it has been pointed out that the kind of animal on which they are made has much to do with the result. Rabbits and guinea-pigs are particularly prone to tuberculosis. Yet, as regards inoculation with tubercular matter, it has also succeeded on other animals, as in the experiments of Böllinger on goats.[1]

Another group of experiments must be alluded to,—those in which pulverized tubercle and cheesy matter have been forced into the lung by inhalation. These have been followed by tubercular-looking nodules; and so have, Schottelius[2] has recently demonstrated, inhalations with non-tubercular substances, such as pulverized calf's brain and cheese, produced apparently identical bodies. They are the result of inflammatory irritation. But microscopically examined, they do not present the appearances of tubercle.

Summing up all the experimental observations, they seem to me to prove that tubercle may be transmitted by inoculation either of true tubercular matter or so-called caseous pneumonia; that resorption of tubercle and infection of previously healthy parts is a view sustained by evidence; that the production of tubercle from non-tubercular material, either by inoculation or by inhalation, is not proved.

[1] Mittheilungen aus dem Pathologischen Institute zu München, 1878.

[2] Virchow's Archiv, lxxiii. 1878.

Inflammatory nodules arise, but they have not the structure of tubercular formations.

I shall now attempt to answer the question what is the exact nature of those inflammatory changes in the lung which give rise to the destructive consolidation, supposed to be non-tubercular, yet from which, by infection, tubercle may come. In other words, I shall endeavor to describe the histology of so-called "caseous pneumonia," or pneumonic phthisis. We find within the alveoli an accumulation of large cellular elements, mixed with leucocytes and exudation matter. We observe the alveolar walls thickened and infiltrated with cells, the vessels compressed, accounting for the breaking down of the bloodless masses accumulated in and around the alveoli. We meet with inflammatory infiltration in the walls of the bronchi, and, as Rindfleisch has so well pointed out, around them, as well as with increased connective tissue between the lobules and around the finer bronchial tubes. In studying the cellular masses we encounter the so-called giant cells. There is, indeed, nothing we do not find in this caseous pneumonia that we have not spoken of in tubercle, only the proportions are different and the admixture of the elements of inflammation more marked. These changes spread usually over a large portion of the lung, and especially as regards the amount of connective tissue, are modified by the duration of the disease. One of the most striking of the lesions, and one which I have rarely failed to encounter in the many specimens examined, is the infiltration with small cells of the walls of the alveoli. Green,[1] too, regards them as very important, and Wilson Fox[2] looks upon them even as tubercular. As the cheesy matter degenerates, evidences of broken-down tissue, with considerable granular detritus, meet the eye. Yet Cohnheim[3] has recently told us that the cheesy part contains in reality but little fat.

Now, is there anything in all this which broadly separates this so-called caseous pneumonia in its minute structure from tubercle? Is there anything more than the evident admixture of a marked inflammatory lesion? Is there anything in the low vitality of the mass and its tendency to decay and fall asunder which is different? And if we call this affection at once "tubercular pneumonia," we are, I verily believe, much nearer to the truth than in endeavoring to separate it from tubercle altogether.

[1] Pathology of Pulmonary Consumption, 1878.

[2] Transactions of Pathological Society of London, 1873.

[3] Die Tuberkulose vom Standpunkte der Infektionslehre, 1879.

But it is not simply on histological grounds that I arrive at this conclusion. I have long studied the subject clinically, and I can record it here as my deliberate opinion that the number of cases of consumption which are supposed to have inflammatory beginnings is grossly exaggerated. They are the exception, not the rule; and even in the cases in which we have had evidence of an active bronchitis or pneumonic condition having seemingly been the start of all the difficulty, how often do we not find, on close analysis, that failing health, hacking cough, even slight spitting of blood, have preceded the acute symptoms? Then, too, we may get the history of inherited scrofulous or tuberculous diathesis. But I do not wish to be misunderstood. There are cases in which none of these qualifying elements can be discerned, which have, to all appearance, started in an acute inflammatory process. It is only the relative frequency of these cases that I am denying.

Again, I ask, what becomes of the instances of so-called pneumonic phthisis? Do they not become tubercular? How many autopsies can any one recall, where persons dying from some intercurrent affection, while laboring under so-called pneumonic phthisis, did not show at some portion of the lung, or in the other lung, miliary tubercle or larger masses which everybody would pronounce undoubted tubercle?

Now, admitting the connection of so-called "caseous pneumonia" or "pneumonic phthisis" with the subsequent development of tubercle,—and nobody denies this, whatever his views as to the character of the connection,—I believe that it is quite as logical to reason from the after-appearance of the tubercle as to the primary character of the so-called inflammation, as to reason from the inflammation and the absorption of its products to the formation of the tubercle. The reasoning backward is as good as the reasoning forward, and, I think, infinitely more likely to be true.

Again, how many cases of ordinary pneumonia happening in perfectly healthy persons are met with which pass, no matter how, into tubercle? Certainly not many. When it occurs there is generally the history of scrofula or tubercle in the family, the taint. Many of the advocates of the inflammatory origin of tubercle, or of its subsequent development after inflammation, tacitly admit this when they speak of the inflammation as special or specific. If it is special or specific, I say at once it is tubercular,—tubercular either from the onset, or it has become so when it presents the appearance of caseous changes.

I am advocating, then, the view that caseous pneumonia leads to tubercle elsewhere, because it is really tubercle already; and that it is not the products of ordinary inflammation, but the tubercular products, which infect. They may appear with the inflammation or be the result of a special kind of inflammation; that does not affect my argument.

Now, one great difficulty in admitting this argument is, that since the researches of Virchow have familiarized us with the facts we cannot assume all kinds of caseous degeneration as tubercular. We know that such changes may happen in purulent collections, in cancer, and that, microscopically, they present the features of the so-called cheesy degeneration which attends pneumonic phthisis. But is there nowhere else similarity of appearance without identity of meaning? Can we tell every case of cancer, under all circumstances, by its cell-growth alone? Are there no healthy textures in the course of formation that look like it? Does every sarcoma present infallible features at all its stages? Moreover, I have already stated that we very generally, nay, almost constantly, find in the pneumonic lesion much the same elements, mixed with the products of degeneration, which we recognize as tubercular in undoubted tubercle.

I believe, then, that pneumonic phthisis is a tubercular pneumonia, and that the inflammation is tubercular from the outset, or has acquired a tubercular nature by changes in the cell-life which we do not understand. Perhaps these are connected with sluggish tissue-change under the influence of a virus,—a taint inborn, or acquired by impure air and bad hygienic surroundings. That we cannot see these things in the protoplasm or cells with even our highest powers is no proof of their non-existence. Do we perceive the manly form of the athlete in the little spermatozoon? What do we find in the ovum to explain the transmission of the delicate features and matchless figure of one generation to the famed beauty of another? Where are, in either germs, the lurking tendencies to disease which we see constantly reproduced in families? Where the specks that indicate cancer, scrofula, tubercle, gout?

The tubercular inflammation may appear as such, and then we have a more or less acute character of case; or the tubercular action may not start for years afterwards. I shall show you some drawings taken from cases that I had watched for years.

Here is one from a woman of 45, who, under my observation, had for eight years a chronically consolidated lung, non-tubercular. Sor-

row overtook her, her general health failed; struggles with poverty came, and she became tubercular. You perceive here how the lower lobe of one lung had undergone the caseous change and contained tubercles, while the upper is simply dark with pigment and densely consolidated. A few scattered, comparatively recent miliary tubercles are found in the other lung.

Here is a yet more striking instance, where a man had for five years a lung which you see, looks exactly like the red hepatization of ordinary pneumonia. Softening, without a vestige of tubercle, is occurring in parts. In a streak at the upper lobe tubercular pneumonia is evident; the other lung is entirely healthy.

Perhaps the views here advocated may appear to call in doubt the transmission of tubercle by resorption and infection,—its constant reproduction, as it were, and scattering through the system. But they do not. These observations are among the best sustained and most valuable in modern pathology, and they are all the easier to understand if we admit the starting-point of the infection to have been a tubercular inflammation. As a contribution to this doctrine of infection and absorption, and also as furnishing many points of analogy with the instances mentioned where inflammation of the lung has been followed by tubercular formations, let me show the remarkable specimens of abdominal tumors and tuberculosis on the table, taken from cases of mine that happened some years since at the Pennsylvania Hospital.

In the first case, occurring in a man the subject of syphilis, and much tormented with abdominal pains, a hard tumor was discovered in the right iliac fossa. This was followed, after some months, by marked emaciation, sweats, diffused abdominal tenderness, diarrhœa, and signs of deposit in the lungs. At the autopsy the mass you see here of dense fibrous tissue was found in the right iliac fossa, below the head of the colon; a small cavity containing pus was detected in its interior; on its exterior were tubercular nodules. The intestines, on their peritoneal surface, throughout their length were thickly studded with tubercular nodules; the mesenteric glands were enlarged and softened; there were miliary tubercles in the lungs.

In the second case, a man also broken down by syphilis, there was the same history of colics and cramps, and a tumor was discovered in the right iliac fossa, three inches from the crest of the ilium. He had noticed the tumor for years. Gastric irritability, tenderness over the abdomen, ascites, diarrhœa, fever, cough, signs of lung-consolidation, became gradually prominent symptoms, and he died exhausted. A

thick, firm mass of inflammatory matter was found covering the cæcum, and had occasioned the tumor. At one part, on the outer and lower wall of the cæcum, was a small cavity containing gelatinoid matter mixed with black, thin fluid. The ileum above the ileo-cæcal valve, as well as the inflammatory, hard tissue in this region, was covered with tubercles. The kidneys contained tubercles; in the left supra-renal capsules were several tubercular nodules. The lungs were full of small, gray, tubercular granulations.

Here, then, are two cases of strange similarity, in which a local inflammation in the abdomen was followed in time by diffused tubercle, both abdominal and pulmonary. But let us return to what I more particularly meant to discuss,—the formation of tubercle in the lungs, and its bearing on the different kinds of phthisis, especially pneumonic phthisis.

Will the views which I have endeavored to bring before you do away with the clinical distinctions concerning which we hear now so much? Not at all. If the so-called pneumonic phthisis has a separate history, gives points for special diagnosis and prognosis, requires different treatment, it ought, no matter what our views of its real pathology, to be recognized as a distinct form. But let us go no further; we are leaving the broad highway for rough and tortuous paths when, as is the tendency of the present day, we attempt to sweep all cases of lung-destruction into the phthisical category.

What do we gain by reverting to a very old nomenclature, and by making phthisis a generic term? To carry this out, we ought to even abolish the word cancer, and speak of it as cancerous phthisis, for surely there is also destruction enough of the lung in pulmonary cancer. The consequence of all this agitation about the word is nothing but confusion. Scarcely two persons seem to use phthisis in the same sense. There are forms of lung-destruction which are clearly non-tubercular; let us give them their special names, and not apply to them a term which has long been applied to that by far most common form of lung-destruction,—phthisis from tubercle. Pneumonic phthisis may be made a separate variety; but that ought to be all. Nay, I venture to suggest whether, in view of the idea long associated with the name of phthisis, and of what I still humbly believe is beyond comparison the most frequent cause of the consumption, it will not be better, unless some other form is particularized, still to retain the name for the tubercular malady, and so to understand it.

But I have claimed your indulgence long enough, and in bringing

these remarks to a close, I can only hope that some train of thought may have been suggested by them which your labors will brilliantly elucidate. *E scintilla lux;* from the flickering glimmer of to-night I trust through others for the steady glow on a subject that sadly needs light.

Dr. Nancrede said that in this disease there was an important series of facts not yet adverted to, but which had an important bearing upon its pathology. In any inflammation of the lungs there is more or less of a cellular infiltration. In certain individuals an inflammation of any organ—*par excellence*, the lungs—results in an exudate composed chiefly of *cells*, while in others the effusion is mostly non-corpuscular. Now, the only way in which solid tissue, like cells, can be absorbed is by fatty degeneration and gradual breaking down—in other words, *caseation*. This conduces to certain evil results, and primarily to the destruction of the infiltrated lung, or other tissue, which is so crowded with cells that its blood-supply is mechanically cut off. Secondarily the broken-down detritus is absorbed, resulting in the deposit of gray miliary tubercle. In those constitutions where inflammation results in an enormous cell-proliferation we are pleased to call them scrofulous or tuberculous, but in reality there is nothing specific in the cause or result of the inflammation. The tendency to cell-proliferation, which must caseate from deficient blood-supply and destroy the involved tissue, is not specific. In the same kind of constitution a very slight blow or twist of a joint will often end in a similar chronic destructive articular disease, while the severest injury to an average patient will result in an acute inflammatory *serous* effusion, which is readily absorbed. Now, no one maintains that the *blow* is a specific irritant. Just so is it with the lungs. Let any simple irritant cause inflammation in one class of cases, and effusions readily absorbable result. Let the same exciting cause act in another set of patients, and an unabsorbable, caseating exudate will ensue. This condition of system may or may not be inherited, for anything that lowers the vital powers tends to lessen its ability to remove the broken-down detritus. More facts and arguments might be adduced in support of this view, but sufficient has been said to indicate the direction in which our observations and researches should be pushed to arrive at a clear and correct view of this obscure subject.

Dr. Tyson said the phenomena of catarrhal pneumonia in children, as compared with that in adults, bore upon the relation of the caseous process to tuberculosis. Most pneumonias in children are catarrhal,

and recovery from them is the rule, while the same lesion in adults frequently terminates in phthisis. If the two processes were the same, phthisis would be a more frequent termination in children.

Dr. Tyson thought the point referred to by Dr. Nancrede—the tendency to the formation of cellular deposits in certain persons generally said to be of scrofulous diathesis—was an important one. It is in cases where the cellular deposit persists and cannot be dislodged that tuberculosis results. Where the proportion of cells in the inflammatory process is small, and they are readily gotten rid of, tuberculosis does not ensue.

Dr. Longstreth said it was true that in the vast majority of lung specimens affected with phthisis it was not possible, either from the macroscopic or microscopic appearances, to determine with certainty whether the disease originated as a tubercular deposit, followed by catarrhal pneumonia and destruction of the lung tissues, or whether catarrhal disease was the initial step. Our inability to decide is due to the confused appearances presented, and to the degenerative changes, both of the morbid products and the normal tissues of the lung, which have occurred.

In respect to the recognition and the classification of certain cells as typical of tuberculosis, he thought that we made a great mistake. In days gone by it was customary to describe cortain cells as cancer-cells, looking upon them as cells with a character foreign to any formation found in the normal organism. These cells and those of all other morbid growths have been considered to be foreign entities which have taken up their residence in our bodies, growing and producing destruction by their growth. Such ideas are the continuation of the old notions of pathology, when every disease was considered to be a possession of our bodies either by a devil or a god. At the present time, in accordance with the teachings of physiological pathology, we trace all symptoms and appearances of disease back to the physiological type of growth, and exclude the devil, and even the gods and other foreign entities, from all share in the morbid processes.

So it is with tubercle. In examining the morbid appearances of the lung subject to destructive changes of this order, it is wrong to look for cells of a foreign nature as typical of this disease. The lung is composed normally of bronchial tubes lined with a mucous membrane, of air-vesicles lined with a mucous-membrane epithelium and backed by various tissues of the connective-tissue type, and of bloodvessels, nerves, and lymphatics. And, excepting when a morbid new forma-

tion results from metastasis, all the diseased processes of the lung commence in one or the other of these various structures, and throughout their duration continue to exhibit the changes which are characterized by the physiological type of growth of the individual tissue involved. Of course, in the lung, as in most other parts of the body, but especially in the lung, from the close proximity and the intimate connection of the various component parts, diseased processes taking their rise in one tissue necessarily involve the adjacent parts, and cause them to take on diseased action; but the parts thus involved secondarily exhibit the morbid action belonging to them, and follow the physiological type inherent in them. Thus, in catarrhal pneumonia, while the morbid process, commencing in the bronchial mucous membrane and extending later to the air-vesicles, affects primarily the epithelial structures of the lung principally, yet the connective tissue and other constituent structures are also involved in the inflammatory processes. The diseased action is, however, not something new and foreign to these parts, but is an exaltation of the physiological type of growth and function. No new and foreign cells are developed, and no new or foreign products are secreted. The normal cells grow more rapidly and the normal secretion is more abundant; and as these cells live a rapid and tumultuous existence, so they die a rapid and tumultuous death.

The interdependence of the morbid processes taking place respectively on the epithelial surface, and in the connective tissues of the air-vesicle walls, as shown to exist in catarrhal pneumonia, is paralleled by the reverse action which results when a tubercular deposit has occurred in the connective-tissue of the air-vesicle wall. The presence of such a formation leads to a catarrhal inflammation of the epithelial lining of the air-cells, and from this time forward the appearances presented by these tissues and their morbid products exhibit retrograde destructive changes similar to those found in catarrhal pneumonia.

What the tubercular formation is, and from which tissue—connective, lymphatic, or vascular—it is derived, are uncertainly-answered questions. That tubercle is connected with and is a formation within one or the other of these three named structures is certain, and therefore that it is of connective-tissue origin—for these three structures belong to the connective-tissue type—is also certain. What is the cause of the tubercular formation and what gives rise to its appearance in the connective-tissue type of structures are also unknown. That tubercle makes its appearance in the lung, as well as in other parts of the body, subsequent to and apparently as the result of catarrhal inflammation,

seems to be well established. That the lung is capable of self-infection in this manner is true, and it is just in these cases that one of the greatest difficulties in the pathology of tubercle arises. When we see the two pathological processes going on side by side—the catarrhal pneumonia and the tubercularization—the confusion of appearances is so great, and the generative changes are usually so advanced, that it becomes impossible to decide which morbid process was the prime mover in the phthisical destruction of the lung-tissue.

It seemed to him evident from clinical studies that the catarrhal inflammation which so frequently gives rise to tubercular infection does not always or necessarily have its seat in the respiratory mucous membrane. The digestive canal is equally capable of furnishing the initiative step in the infection, especially so as the disturbance of nutrition, caused by the defective digestion, favors the development of tubercular disease. A chronic catarrhal gastritis, with the attendant pharyngeal irritation, not infrequently is an antecedent phenomenon to a bronchial or pneumonic inflammation of a chronic variety. The diseased process extends from the digestive mucous membrane tract to the respiratory. It is probable that a close investigation and comparison of a large number of cases would show that the kitchen is a large factor in the induction of phthisical destruction of the lung, and that it would be found that the doughnut and pie breakfasts so common in rural New England life have as much to do with lung diseases as the rigor of the climate of that section of the country.

Dr. F. P. Henry said the paper of the evening incidentally demonstrated that the too rigid separation of the clinical study of disease from that of its histology is detrimental to progress. The cases quoted by Dr. Da Costa were of unusual interest and value, from the fact of their careful observation during life, as well as post-mortem. Although pneumonic phthisis is often developed in individuals of apparently robust constitution, it is exceedingly rare for it to occur in those with a well-developed vascular system, evidenced by a strongly-acting heart, and a full, strong pulse.

Much attention has been paid to the lymphatic system by students of the etiology of phthisis, and yet comparatively little is known regarding the lymph circulation and the properties of the lymph-cell. For instance, it is not known whether in stasis of lymph, diapedesis of its cells may occur. Supposing this to take place, it would be tolerably certain that a depot of such immature cells would readily soften and break down. Following the same theoretical line of thought, another

point of inquiry would be, as to whether such cells, as escaped destruction, would have a tendency to the formation of *adenoid* in preference to fibroid tissue.

Certain facts support the view that a sluggish lymph circulation is concerned in the origin of phthisis. It was long ago noticed by Lebert that in cases of stenosis of the pulmonary artery tuberculosis is exceedingly frequent, and it is also generally accepted that there is a certain degree of antagonism between mitral disease of the heart and phthisis. These facts have an important bearing upon the view that a sluggish lymph circulation is of influence in the production of pneumonic phthisis. The lymph circulation is but a continuance of that of the blood, and is therefore largely dependent upon the heart. In stenosis of the pulmonary artery stasis of lymph is to be looked for, whereas in mitral disease, the vessels of the lung being engorged with blood, the pressure upon the lymph-spaces and vessels would be a hindrance to the migration of lymph-cells, supposing such to occur under any circumstances. As the lymph-cells possess the amœboid movement, it is, to say the least, not unreasonable to suppose that they may be possessed of other properties of the white blood-cells. The study of the lympathic circulation in connection with the etiology of phthisis is yet in its infancy.

Dr. Eskridge said he did not think that most cases of phthisis were due to a bad diet. Clinical experience teaches that in catarrhal phthisis the digestion is good. He had seen many cases where the lung was consolidated and the digestive organs had remained healthy. But in tuberculosis the digestion is faulty, yet symptoms of tubercle have preceded the digestive troubles. Dr. Eskridge thought most cases of tubercle were due to heredity. There is no reason why taints should not be transmitted. Experiment has shown the possibility of artificially-induced epilepsy becoming hereditary. If certain cells are affected in the parent, why should they not be so charged in the offspring? If healthy parents are necessary for healthy children, is the opposite less true? *April 22d*, 1880.

### 3. *Differential diagnosis of cerebral embolism and thrombosis in cases of disease of the valves of the heart.*[1]

Read by Dr. C. K. Mills.

Dr. Charles K. Mills said that some points in Dr. Musser's notes recalled a case which he had recently examined post-mortem, and in connection with which he wished to make some remarks on the differential diagnosis of cerebral embolism and thrombosis in cases of valvular disease. The patient was a woman more than sixty years of age. During life he had made the diagnosis of fatty degeneration of the heart, and of aortic, and probably mitral, disease. Dr. Bruen, who saw the case in consultation, concluded that only the aortic valves were diseased. The radial and temporal arteries were markedly atheromatous. She had had three attacks of right-sided paresis or paralysis, dying after the last, which came on suddenly. Post-mortem examination showed extensive calcification of the aortic crescents and degeneration of the heart-walls. The aorta and the cerebral vessels, both large and small, were atheromatous. Centres of softening were found in the motor zone of the cortex of the brain, and also in other regions. Several of the secondary and tertiary branches of the middle cerebral artery were found to be closed, the occlusion being due to disease of the walls of the vessels and the formation of thrombi. In cases like this the diagnosis of embolism is not infrequently made, although the condition found is really thrombosis. He would give the following as good diagnostic points. In favor of thrombosis would be (1) advanced age; (2) evidences of atheroma of vessels; (3) fatty degeneration of heart; (4) a succession of slight attacks of paresis. In favor of embolism would be (1) youth; (2) absence of signs of atheroma; (3) previous history of rheumatism; (4) a comparatively severe attack of paralysis.

He, of course, did not mean to go into a detailed discussion of embolism and thrombosis, but only to call attention to one or two differential features.

Dr. Tyson asked if the urine had been examined for sugar.

Dr. Musser, in reply, stated that appropriate tests had been applied, but with negative results.

Dr. Tyson, in explanation of his question, said that we have here

[1] Remarks made in connection with Dr. Musser's specimens, presented Nov. 11th, 1880. See p. 95.

hypertrophy of the pancreas—a condition which Niemeyer, on the supposition that diabetes is due to the presence of an increased amount of pancreatic ferment, says always attends diabetes. This is only one of many instances constantly occurring, which show that such a theory of diabetes is inapplicable—at least to most cases.

Dr. Henry remarked, in reply to Dr. Mills, that his experience had led him to believe that softening of a portion of brain-tissue might occur without complete occlusion of the vessels supplying it. About three years ago he was summoned, early in the morning, to an old gentleman who had awakened to find a loss of power in his left hand. While he was examining him, the paralysis gradually passed away and returned, and in the course of an hour this intermittent paralysis occurred several times. There was no abnormal cardiac sound, and the kidneys, from previous examination, were known to be healthy. On further study of the case, it was noticed that the paralysis, which continued in this intermittent manner for several weeks, was always at its worst in the early morning, when the heart was acting most feebly. The superficial arteries were not at all atheromatous, so far as could be decided by the touch; nevertheless, the diagnosis of partially occluded cerebral vessels, with a slightly fatty heart, was confidently made. The treatment completely confirmed this theory. A few teaspoonfuls of wine taken before rising in the morning caused an immediate improvement in the morning attacks, and under the continued use of the iodide of potassium in small doses the paralysis disappeared. In about a year a second, more decided attack occurred, this time involving the facial muscles of the left side and, in a slight degree, the left leg. This attack was also recovered from, to a great extent, the only perceptible trace remaining being a certain slowness in pronouncing particular words and a slightly diminished grasp of the left hand. A third attack in June last proved fatal—about six weeks from its commencement. Its onset was more sudden than either of the two former, but by no means *foudroyant*, and it was accompanied by decided symptoms of cerebral softening. At the same time Dr. Henry had under his care a hospital patient, a woman aged about seventy, whose case was almost identical with the one just outlined, even to the time of the attack and the date of death. An autopsy was obtained, and a softened mass, almost diffluent at its centre, was found occupying the extraventricular portion of the right corpus striatum. The small arteries supplying this portion of the brain (arteriæ corporis striati) were traced from their origin—from the middle cerebral artery—to the soft-

ened portion of brain, and, although decidedly atheromatous, were not occluded at any point.

Dr. Longstreth said that he agreed with the remarks of Dr. Mills as to the distinctive points in the differential diagnosis of cerebral embolism and thrombosis, so far as mentioned by that gentleman. He was surprised that one very important differential point had been omitted,—viz., the suddenness of the invasion of the symptoms in embolism in contradistinction to the gradual onset in thrombosis. In cases of embolism, a vegetation becomes detached from the diseased heart-valve, and is carried by the blood-current into one of the cerebral arteries. The immediate effect is to cut off the supply of blood to the vascular area beyond the point where the embolic mass is lodged, and that portion of the brain becomes functionally dead. This process is a rapid one, and, if the area involved corresponds to the motor ganglion, hemiplegia results with a suddenness comparable to that due to cerebral hemorrhage. In thrombosis, on the other hand, the closure of the vessels is effected by a gradual process.

Dr. Mills could not agree with Dr. Henry if he meant to teach that extensive softening, or, in fact, any softening of the brain could occur without closure of vessels. Softening of the brain was a process altogether peculiar,—a necrobiosis. In order that the pathological process shall result, vessels somewhere must be *closed*. The vessels may be large, may be of medium size, or may be very small, but occluded they must be before softening can take place. We never find true cerebral softening without complete blocking of some of the vessels going to the part. He even doubted the existence of the so-called inflammatory softening of the brain, except in so far as processes of inflammation might lead to occlusion of vessels. He made this assertion knowing that one school of pathologists held a different opinion. In the case related by Dr. Henry, vessels were found diseased, and their calibre diminished, but they were patulous. May not some occluded vessels have escaped observation? Even with the greatest care, such as was no doubt taken by Dr. Henry, diseased vessels will sometimes elude us. Numerous nutrient vessels, branches both of the anterior and the middle cerebral vessels, supply the corpora striata. These too are terminal vessels, each supplying a limited area, and not having anastomosing branches. Some of them might readily, in the most careful hands, be broken, torn, displaced, or overlooked. He thought that *hardening* and atrophy of the brain were more likely to result than softening from a poor supply of blood or

from a supply of poor blood. He was not without the weight of great authority—that of Hughlings-Jackson, for instance—for such views as he had tried to advance. In connection with a discussion of this kind, the fact that necrobiosis may result from numerous occlusions of cerebral capillaries should be remembered.

Dr. Mills considered the point made by Dr. Longstreth, that paralysis from thrombosis comes on slowly, and that from embolism suddenly, a very good one. He should not have omitted it in his enumeration of differential points. He did not, however, look upon it as absolutely distinctive. Some cases of cerebral thrombosis do at times develop paralytic symptoms suddenly. The degenerative disease of the vessels proceeds slowly, the narrowing of the calibre of the vessel taking place gradually; but the final act of closure, the completion of the blocking, may be practically sudden, and thus give rise to the same shock as occurs in a case of embolism. He might here take occasion to remark that embolism and thrombosis do not *always* induce softening. In a brain examined by Dr. Shakspeare and himself, they had found twenty or more points of closure, but only seven foci of softening in the convolutions. The escape from softening in such cases was due to collateral circulation, which in the cerebral cortex was freer than Duret would admit.

Dr. Henry agreed entirely with Dr. Mills in regard to the necessity of care in the post-mortem examination of the brain. His experience as physician to a large hospital had long ago taught him this fact, not only in connection with the brain, but also in regard to every other organ of the body. As concerns the case in question, the centre of cerebral softening having been found, the vessels had been traced from it to their point of origin from the middle cerebral artery. No difficulty whatever had been encountered in making this dissection, the softened brain-tissue being readily and completely removed from the small though rigid bloodvessels. Numerous patches of atheroma were observed in the walls of these vessels, but their lumina were patulous. Dr. Henry thought that a weak heart, in connection with but partially occluded vessels, might lead to cerebral softening. He could on no other theory account for the symptoms of the case to which he had first referred, in which the paralysis was intermittent, in the course of an hour a complete paralysis being followed by an almost complete restoration of power, and this on numerous occasions. Remedies which strengthened the action of the heart, such as wine, caused an immediate improvement in the symptoms; and, conversely, the

paralysis was always at its worst when the patient had been the longest deprived of food, and the heart in consequence was acting most feebly,—namely, in the early morning before breakfast. Finally, the symptoms of cerebral softening were completely developed in subsequent attacks. Taking this case in connection with the almost precisely similar one in which an autopsy was obtained, and in which the most careful possible search detected no vascular occlusion, Dr. Henry was so firmly convinced of the possibility of cerebral softening without complete vascular obstruction as not to be shaken in his belief by merely negative evidence.

Dr. Longstreth said that he thought it was supposable that a sudden closure of a large vessel was possible without embolism ; for example, a cerebral artery where the diseased inner coat suddenly became detached and occluded the calibre of the vessel. Again, a rapidly occurring hemiplegia or unconsciousness in the absence of valvular heart disease might possibly be due to thrombosis, provided the occurrence of a cerebral hemorrhage could be excluded. Cerebral embolism did not occur except in connection with endocardiac or valvular disease, or with atheromatous changes in the large vessels. Sudden hemiplegia did not occur except from cerebral hemorrhage, embolism, or possibly, as in the present discussion, from thrombosis, unless symptoms of other cerebral diseases pre-existed ; for example, tumors. If heart or vascular disease were absent, embolism could be excluded. In all cases of sudden unconsciousness, therefore, it was a question between cerebral hemorrhage and thrombosis. Several years ago, Dr. Longstreth said, when examining a number of cases of cerebral embolism at the Pennsylvania Hospital, he had consulted the records of medical literature concerning this question, and had not been able to find any well-authenticated cases of cerebral thrombosis. In his personal experience the cases of thrombosis all originated from the sinuses or were of venous character ; in these cases, of which he had examined several, the cerebral symptoms were sudden in their onset. Hemiplegia or unconsciousness did not characterize them, although paralysis developed subsequently.

He considered Dr. Mills's language was too weak a characterization of this symptom. The point in regard to the suddenness of the symptom was not only a *good* one, but a *capital* one. Dr. Longstreth said he would like to ask for Dr. Tyson's experience in the matter.

Dr. Tyson agreed with him, but could imagine the possibility of the occurrence of cases such as those mentioned by Dr. Mills.

Dr. Longstreth then asked whether Dr. Tyson knew of any such recorded cases.

Dr. Tyson said that he did not.

Dr. Mills said that they were both theoretically and practically possible, and he thought that observers had overlooked this fact.

Dr. Longstreth could not see how any condition of vessels could result, such as would lead to complete and sudden closure of a cerebral vessel, without precedent cerebral symptoms having been present, either in their slighter form, as mental feebleness or drowsiness, or in their graver form, such as is due to cerebral syphilis. Until proof of such cases having been observed were adduced, he should continue to doubt their occurrence.

Dr. Mills said that he had seen more than one patient stricken with paralysis suddenly when he also believed thrombosis was the morbid condition present. He had several times confirmed his diagnosis by autopsies. The case reported by him this evening was one of this kind. Usually, however, prodromata are exhibited in these cases.

Dr. Tyson thought that of course this would be the only proof of Dr. Mills's theory, and hoped that he would at a future time report the cases referred to, upon which he founded the statements that he had made this evening.

Dr. Shakespeare said that, since the field of theory had been entered upon, he could conceive of cases where spasms of the vessels from reflex causes might produce sudden unconsciousness, which would, of course, disappear in due time. *November 11th*, 1880.

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### 4. *The etiology of tumors.*

Read by Dr. H. F. Formad.

The etiology of tumors is a dark chapter in pathology. I seized with great pleasure the opportunity to read a paper on this topic, as it concerns questions in which many of us are deeply interested. During the last few years I have spent a great deal of time in the study of the cause of tumors, but, as will be seen, with only moderate success. It is to the discussion of this subject by the members of this Society and to the criticism which is to follow that I look forward with deep interest for practical points, information, and instruction.

Not only is the etiology of tumors insufficiently treated of in the

respective text-books, but the literature of the subject is not generally accessible, hence I think it will be a welcome addition to American medical literature if I represent each and all of the various author's views concisely, but fully enough to avoid misrepresentations. Then follow all the experimental researches concerning the inoculability and origin of tumors, including researches made by myself and other students of pathology in the pathological laboratory of the University of Pennsylvania.

I. Views of Authors.—We shall first have to consider what new formations authors include under the heading of tumors, inasmuch as this stands in direct relation to their view of their etiology. Much less discrepancy between the views will be observed if we define this more closely.

In the sense of Virchow, any circumscribed elevation over a given surface or any excessive enlargement is considered a tumor. The products of specific inflammation such as tubercle, gumma, glanders, lupus, and lepra, also the cysts and most of the hypertrophies would consequently belong here. Those authors who regard the products of specific inflammation as tumors, naturally draw from this the inference that most of the tumors are the result of inflammation. Other authors exclude from the category of tumors the above-mentioned inflammatory products, together with cysts and hypertrophies, and hence they class with tumors only new formations, for which an evident cause had not been proven, explaining the etiology of the latter by all kinds of hypotheses.

Authors change their views occasionally, and some now take a stand in the question of the histogenesis and etiology of tumors different from that expressed in previous writings. I have been careful to represent the doctrines as they stand to-day.

The different authors and their views of the etiology of tumors can conveniently be classed in the following five groups :—[1]

1. Predisposition and inflammatory theory: Virchow, S. D. Gross, Woodward, Samuel, Wagner, Birch-Hirschfeld, Cornil and Ranvier, Perls, Tyson, S. W. Gross, Fitz.[2]

2. Dyscrasia theory: Rokitansky, Paget, Billroth, Simon.

[1] I enumerate here only those authors who, to my knowledge, have expressed themselves more or less definitely on this particular subject.

[2] This grouping of authors concerns only their views on etiology; those on histogenesis would require different grouping.

3. Embryonal theory: Cohnheim, Thiersch, Waldeyer, Lücke, Maas, Hasse, Epstein.

4. Idiopathic or spontaneous theory: Rindfleisch, Stricker, Nancrede, Payne.

5. Nervous theory: Van der Kolk, Lang, Snow.

1. *The Predisposition and Inflammatory Theory.*—The idea of regarding tumors as inflammatory products is not new. John Burns[1] described medullary cancer as a sponge-like inflammation. The French school in the early part of this century, particularly Broussais[2] (1826), regarded all tumors as products of chronic inflammation. More than this, even Galen[3] in the second century, 1700 years ago, says that "nasal polyps are due either to inflammation or develop from a node (Phyma) or from germinal matter (Blastema)." Virchow says that many tumors are undoubtedly the products of inflammation, and that it is difficult to draw a line separating them from those tumors whose origin cannot be ascribed to inflammation.

In the etiology of tumors there are after Virchow to be considered three causal conditions: first, local causes upon which depend the development of a tumor in a particular place; second, a general predisposition; third, a general cause, which, for the sake of convenience, is made to relate to the fluids of the body, and is called dyscrasia or cachexia. "Cachexia," Virchow says, "is not always present. It never has been observed in the beginning of the formation of the tumor, but always as subsequent to it, and the condition can be brought about by other than cancerous juices. Cachexia will manifest itself only in such persons in whom the stomach, liver, or lymphatic apparatus becomes primarily or secondarily affected by cancer or anything else. There is a physiological predisposition in certain tissues to be more readily attacked and sooner affected by disease than others. Highly organized tissues are very little predisposed to excessive abnormal formative activity. The latter occurs more in the connective tisue and its derivatives, and the lymphoid and epithelial tissue; in the epithelial tissues the new formation takes its departure usually only from the younger undeveloped cells (*i. e.*, from the lower stratum of the *rete mucosum*). This predisposition in tissues may be acquired, it does not need to be inherited.

[1] Dissertation on Inflammation, Glasgow, 1800.

[2] Broussais, Histoire des Phlegmasies Chroniques, Paris, 1826.

[3] De tumoribus praeter naturam. Cap. 17, quoted after Virchow, Die Kr. Geschwülste, I., p. 35.

"In opposition to the humoro-pathological doctrine is brought the neuro-pathological, by which several enthusiastic neuro-pathologists sought to explain neoplastic formations. These authorities cite such causes as care, distress, lesions of nerves, and serious febrile disturbances. These causes are mostly brought forward in case of cancer of the stomach; but the arguments are very weak. Statistics show that cancer, and especially cancer of the stomach, occurs more often in the higher, wealthier classes than among the poor. The nervous symptoms are caused by the presence of the cancer. In cases of all tumors the nerves may, on account of weakness and disturbance of nutrition, be a predisposing cause and no more. . . . .

"Not being able to find the cause of tumors in the blood or nervous system, we naturally turn to the tissues themselves, from which tumors proceed. An important fact to be mentioned in this connection is a local disposition to form tumors. In addition to this the hereditary transmission is unquestionable.

"The hereditary epidermal growths and fibromata are tumors developing directly from certain tissues, and do not suggest any dyscrasia foundation.

"The other members of the class of hereditary growths make their appearance after birth at longer or shorter intervals. To this class belong the hereditary growths of the mammæ, uterus, skin, stomach, lymphatic glands, and lung, which are often seen in many generations of the same family; *e. g.*, tuberculosis, syphilis, cancer, etc. But these are only predisposing causes. It is the predisposition which is inherited, not the disease.

"It is, however, certain that these growths appear at certain periods, such as the menopause or the beginning of menstruation; in such cases we must conclude that there exists in the tissues a certain weakness by which they are unable to adapt themselves to the disturbance which certain changes in the life processes entail, and that there is undoubtedly some organic condition or incomplete development. This condition is illustrated by the fact that certain tumors (keloids, etc.) recur in the same cicatrix, and that a cicatrix is generally a tissue not mature, and cannot compare in point of completion with the tissue which it ought to replace. Nævi are congenital irregularities, and at later periods can develop into malignant growths. . . . .

"We also know that on mucous surfaces tumors occur for the most part in such places where there previously was a simple inflammatory disturbance; *e. g.*, where the simple inflammatory hyperplasia of chronic catarrh precedes the growth of polyps, and polyps may develop into can-

cer. In cancer of the stomach there is always seen a gradual transition of the catarrhal products to carcinomatous structure. Exostoses, warts, elephantiasis, are all preceded by inflammation. We next come to certain regions of the body which, from their situation and the character of their function, are exposed to irritation and injuries. This renders them especially predisposed to take on a diseased nature; *e. g.*, the stomach, sexual organs, bones, skin, and the different orifices. If we make an estimate of all malignant growths we find that most occur at orifices and at the orifices which are exposed to the greatest injury, consequently those of the digestive and sexual apparatus are most affected.

"We know that certain organs are peculiarly predisposed to take on certain diseases, for, in following the history of a tumor we must come to the conclusion that in the tissue where the tumor exists there was some local predisposition. We know that not all kinds of tumors can develop in all organs. They have their favorite seats, certain tumors are seldom found in certain organs, while tumors found in one organ are seldom found in another. There must be something in the normal anatomical build which determines its mode of development. The same is true of secondary growths. In the testicle we frequently find secondary tubercles, but very seldom secondary carcinoma. We may say that nearly all those organs which are especially predisposed to primary growths, exhibit very little predisposition to metastasis; conversely we find metastasis most frequently in the lungs, liver, kidney, and serous membranes, and just these parts are least prone to become seats of primary growths. The skin, mucous membrane, eye, nose, and testicle are the common seats of primary growths and are seldom the seats of metastatic growths. The lymphatic glands, brain, muscle, and bones stand between the two groups. The lymphatic glands are prone to metastasis and also to primary growths, bone, to primary growths, being frequently the seat, also, of metastasis." All these facts together lead Virchow to the conclusion that even in malignant growths the primary growth does not come from a dyscrasia, and that every tumor is local and frequently of inflammatory origin.

These are the doctrines generally adopted, doctrines for which Virchow alone receives credit; but if we look through the older literature, and even through the native literature of America, we shall find a book written twenty years before Virchow's time, in which those fundamental ideas concerning tumors are set forth in all their essential points. I have reference to Prof. S. D. Gross's work, *The Elements of Pathological Anatomy*, published in 1839. I shall quote only a

few sentences. "Tubercle," Prof. Gross says, "is always the result of inflammation, and that this is the case likewise with scirrhus seems sufficiently evident from what has been stated in regard to its exciting causes. Very frequently, it is true, the disease arises imperceptibly without local injury or obvious constitutional derangement. But this certainly does not prove that inflammation is not concerned in its production. How often do we find traces of inflammation after death, without having had the slightest indication of it during life? The fact, then, that inflammation is not manifested always by the usual phenomena, does not invalidate the idea of its presence." In another place he says: "Predisposition must also be accounted for, and, in some instances, it seems to be connected with an hereditary taint, being transmitted from parents to their offspring." Hence, he believed in the local inflammatory origin of tumors and conditions which predispose to them.

Dr. Woodward, U. S. A., in his excellent paper on the structure of cancerous tumors,[1] agrees also with Virchow, saying: "The origin of the first growth was always to be looked for in local influences. Former injuries of one kind or another could be affirmed in a large number of cases to have preceded the development of the disease, and, though frequently the patients had lost all recollection of the original harm, yet it was in this direction we ought to look rather than seek to explain away the real difficulty by invoking the aid of an imaginary cachexia."

Although Dr. Woodward at another page says: "The time has not come yet for any one to tell why cancer originates," he must be classed with the authors of the inflammatory theory of tumor formation, as he expresses the view that cancer cylinders are largely formed from migrated white-blood corpuscles; the latter when infiltrating tissues being always of inflammatory origin.

Samuel, of Konigsberg,[2] although agreeing with Virchow in all the essential points, maintains more strongly than anybody else the view that inflammation is the main cause of tumors. He says: "The idea of excluding or even limiting the causative relation of chronic inflammation to neoplasms leads far astray from the right path."

The main reason for this view of Samuel is obvious. Like Virchow, he cannot conceive of the difference between permanent inflammatory products and tumors, and he classes the products of specific inflammation, *e. g.*, gumma, tubercle, lupus, etc., with the tumors. He does not admit, however, any special predisposition, nor does he admit Cohn-

[1] Toner Lectures, Washington, 1873. [2] Handbuch d. Alleg. Path., 1879.

heim's embryonic misplaced or supernumerary cells,[1] but simply proliferation of cells by a process similar to chronic inflammation; a mere intensified afflux of blood he does not think sufficient.

The formation of malignant tumors capable of metastasis depends, according to Samuel, upon the want of resistance of the surrounding tissues or the removal of resistance of a given normal boundary. The formation of all tumors he ascribes to mechanical or chemical irritation, direct disturbance, "tearing, stretching" of tissues. Tissue is formed above physiological want and limit. The consumption being overbalanced by the proliferation of cells, "from the excess of new-formed material arises the tumor."

Wagner, Birch-Hirschfeld, Cornil and Ranvier, and Perls hold the views as propagated by Virchow in their essential parts. Of our American pathologists and writers on this subject, I think, we must include here Prof. Fitz, of Boston, and Prof. Tyson and Dr. S. W. Gross, of Philadelphia.

2. *Dyscrasia Theory.*—To avoid misrepresentation I will quote literally all the strong points of the authors of this view.

Rokitansky was the first advocate of this theory in its present state.[2] The ablest advocates of this doctrine, are, however, Billroth and Sir James Paget.

Sir James Paget's views are best expressed in a "Discussion on Cancer," at a meeting of the Pathological Society of London.[3]

He holds that cancer is analogous to syphilis, gout, scrofulosis, and other blood diseases, and he says that the transmission of cancer accords perfectly with what we see in them, not with what we see in local disease. He believes in a constitutional peculiarity residing in the blood, and that any exciting cause, in such a subject, may call forth in him a cancerous growth. Paget says: "While inheritance in simple tumors transmits the tendency to growth in one special part or special tissue, the hereditary tendency to malignant growths is not attached to any one organ or tissue, but may break out anywhere (?). What is transmitted is, then, not a property of any part of the body, but of the body as a whole. . . . .

"I must maintain, therefore, that all the grounds against holding that cancer is a constitutional disease, will hold as strongly against a belief in any disease whatever being a blood disease."

Billroth[4] holds a similar view. He says: "While fully recognizing

[1] See Embryonal Theory.
[2] Rokitansky, Handbuch, 1843.
[3] British Medical Journal, 1874.
[4] Surgical Pathology.

the exercise of observation and brilliant ideas by which it is attempted to prove the purely local disposition to development of tumors, I cannot consider the proof as at all convincing, but remain of the opinion that there is just as much a specific predisposition to the development of tumors as there is to chronic inflammation, with proliferation of the inflammatory new formation, with suppuration, with caseous degeneration, etc."

"I would remark," he says, "that in regard to primary tumors we may assume in many cases that there are also specific, so-called internal irritations developing in the body itself. . . . . .

"For my part I find it just as allowable to assume a diathesis here as in scrofula, arthritis, etc.; that partly from unknown, partly from known causes of general nutrition and ordinary conditions of life, abnormal matters proceed, which have a specific irritant action on this or that part of the body, analogous to that of certain drugs. Lastly, if to this we add that the diathesis for production of tumors is hereditary, although not to such an extent as the chronic inflammatory diathesis, the doctrine of weakness localized in certain systems of tissue, or certain parts of the body, seems entirely untenable. There is certainly a local cause for the members of one family having large noses; in proportion to the face, they have grown larger than in other men, still the large nose of the father cannot descend directly to the son, it can only be inherited from the father through the spermatozoa, and there the original cause is to be sought; all peculiarities that descend by inheritance are unquestionably to be termed constitutional. . .

"I think that the original causes for the local requirements for the development of tumors must be sought in specific peculiarities of the individual organism; in the same way we speak of a scrofulous or tuberculous person, meaning the pathological race, as it were, to which the individual belongs. . . . .

"Tumors, like inflammatory neoplasms, result from irritation of the tissue; the difference in the cause lies: 1. In the specific quality of the irritation. Infection of healthy tissue about a tumor, neighboring lymphatic glands, etc., is considered sufficient proof of this. It is supposed that under some unknown circumstances this specific irritant may be formed locally.—(Rindfleisch.) I think that, partly as a result of hereditary predisposition, partly from a developed tendency, that is, where there is a diathesis we may imagine the formation of materials in the fluids of the body, which shall have specific irritant action on one or other tissue. 2. Any, usually an inflammatory irritation, may

excite a tumor if the irritated tissue is specifically disposed for the development of growths."

As a curiosity I would mention the following view.

John Simon[1] regards a cancer as a gland, as a newly formed secretory organ, which is designed to collect effete material from the juices of the system in the same way as the kidney collects the urea from the blood. If the tumor is opened by ulceration a true excretory organ is created and the dyscrasia is temporarily suspended. Regarding cancer from such a standpoint, extirpation would be just as injurious as extirpation of a kidney. For by extirpation of such an elementary organ the individual would be exposed to danger, as the physiological condition which had been created to relieve the dyscrasia would be destroyed.

The benign tumors Simon classes as monstrosities. Of late, however, Simon[2] has given up the dyscrasia view altogether. However persistently Paget and Billroth may defend their old views, I do not think that any one earnestly believes any more in the dyscrasia theory of even the malignant tumors. Paget himself admits that "cancer is eminently a disease of degenerated tissue."

Virchow properly remarks:[3] "If there should exist primarily a specific dyscrasia we would not have a single, primary tumor focus from which metatasis proceeds, but we would have tumor eruption in all possible places without any definite plan."

3. *Embryonal Theory.*—Cohnheim, of Leipzig,[4] holds rather an exclusive view. Regarding tumors as typical new formations of embryonal origin, he classes them as malformations, forming a subdivision of "monstra per excessum" (monstrosities). In order to understand perfectly this view of the etiology of tumors it will be necessary to present a few details of his doctrine.

Cohnheim excludes from the group of tumors the cystic growths and all the products of simple and of infectious inflammation, as well as the simple hypertrophic products. He retains, however, in the class of tumors the group of dermoid cysts or Virchow's teratoms, using the well-established congenital derivations of the latter as a basis for his hypothesis of the etiology of tumors.

[1] Simon; Text-book on General Pathology, London, 1850.

[2] See Simon: Discussion on Cancer, British Medical Journal, 1874.

[3] Virchow die Kr. Geschwülste, vol. ii. p. 252.

[4] Allegemeine Pathology, 1877.

Cohnheim is the most energetic advocate of the purely congenital origin of tumors, and makes their origin depend upon "an anomaly," or irregularity in the construction of the embryo.

With his usual positiveness in assertions, he most emphatically denies the possibility of any other direct cause of tumors.

Traumatism and the different irritations can, according to him, give rise to hypertrophic and inflammatory products, but not to true tumors. He also denies positively an infectious origin of tumors (the products of specific inflammation being excluded by him) upon the ground that tumors are never communicated from a patient to the surgeon, or from wife to husband; and on the ground of the many fruitless attempts of inoculation from man to animals, or from animal to animal.

He admits, however, that living epithelial cells transplanted into the skin or aqueous humor of an animal will temporarily proliferate, but they will disappear within two weeks by absorption without impairing the health of the animal, the result being an abscess, and, ultimately, a scar.

Small particles of periosteum introduced by Cohnheim under the skin of animals and into the lungs of dogs and chickens, by means of the jugular vein, had a similar fate. The particles of periosteum increased in size, ossified, but ultimately disappeared.

I will have to refer to these experiments again, which, by the way, have been repeated lately by Henry Wile in the pathological laboratory of the University of Pennsylvania, giving entirely different results.

Cohnheim further denies that nervous and other influences stand in a causal relation to tumors. True tumors, according to him, cannot originate by virtue of any kind of interference; only one causal factor exists, viz., an "*anomalous embryonic arrangement.*"

Similar views have been so far expressed by other pathologists in regard to the origin of the dermoid tumors alone. These have been proven by Lücke and others to be due to anomalous invaginations of the outer layer of the blastoderm during the formation of the structure of the eye, mouth, neck, the ovaries, the testicles, etc.

Cohnheim applies to all neoplasms a similar mode of origin. He explains the anomalous embryonic arrangement which forms the starting point, and becomes the cause of tumors, by the following hypothesis:—

He says: "In an early stage of embryonal development there may be undoubtedly produced more cells than are necessary for the construction of a certain part; so that a certain number of cells remain

superfluous. Their number may be very small, but they possess great proliferating power on account of their embryonal nature. This must occur before the complete differentiation of the blastodermic layer and the formation of organs." This appears to him the easiest explanation, why, from such a misplacement, there occurs, not the overgrowth of a certain part of the body, *e. g.*, giant leg, but simply a histoid tumor, *i. e.*, it results in an excessive overgrowth of only one tissue of the part. The superfluous cell material may be distributed uniformly, or it may remain together in one place. In the first case there will result a superfluous part of the system like supernumerary fingers, etc.

"A positive proof," he says, "cannot be given; but there are numbers of facts which render this hypothesis probable in the highest degree."

Here he enumerates inheritance of tumors, quoting the well-known instances of congenital tumors in members of the same family and in different generations; again, the fact that children are frequently born with tumors; referring also to the histogenesis of dermoid cysts. "The congenital tumors form only the minority, and our theory would be a weak one," he says, "if we should base it only on this last fact, but we do not expect always that the individual will be born with a tumor; we only demand the existence of a quantity of cells produced, above physiological requirements, from which the tumor may be developed."

"To promote the production of the tumor we do not need any nervous influences or any kind of excitation, but only and alone *need sufficient blood supply.*"

Cohnheim admits that irritation and traumatism favor the formation of tumors, but that inflammation is by no means necessary for their development, and that no true tumor can be formed unless superfluous cell masses be present. The hypothesis of undeveloped germs is warranted by many facts. The mammary glands, the testis, the ovaries, and the hair follicles on the lips remain latent up to puberty, the latter occasionally, begin to proliferate in women after the menopause.

The extraordinary growth of the muscular fibres of the uterus is referred by Cohnheim to the stimulus given by pregnancy, to cells which before were quiescent, and the frequent occurrence of myoma in the womb of sterile women, to the abnormal development of the same cell masses, when their growth would answer no physiological purpose.

The occasional occurrence of extra fingers and toes proves the exist-

ence of more cells in those individuals than was needed for the normal organism.

All primary heterotopic tumors Cohnheim supposes with Lücke and Waldeyer to be caused by aberrant cell masses; so the adenoma of the axilla, by the development of misplaced cells cut off from the mammary gland; chondroma of the parotid, to those properly belonging to the ear; chondroma and osteoma of lungs to those from the ribs.

Both Cohnheim and Virchow have noticed cartilaginous islands in the shaft of adult bones, which, being according to Cohnheim supernumerary, may be inferred to give rise to the development of chondroma and osteoma commonly met with in these parts.

Cancers are known to occur very commonly at the orifices of the alimentary canal, *e. g.*, on the lower lip, cardiac and pyloric ends of the stomach, rectum, etc. Cohnheim offers an explanation by his hypothesis, to wit, that every such locality has been the seat, in embryonic life, of some anomaly in development, such as the deposit or aberration of surplus cell masses which, if excited by excessive blood supply, will develop into a tumor.

Germs may fail to develop on account of the lack of necessary stimulus, or because of the resistance of surrounding structures.

Epstein[1] is a decided believer in Cohnheim's embryonal hypothesis. Having discovered epithelial pearls in the mucous membrane of the gums, the tongue and the genitals of new-born infants, he thinks that he has found a proof for Cohnheim's view, regarding these pearls as the famous supernumerary embryonic collections of cells. These having no physiological purpose may either disappear, or under certain conditions of nutrition form the starting point for tumors.

Prof. Maas, of Freiberg,[2] declares himself strongly in favor of Cohnheim's theory on the etiology of tumors. He rejects positively all other theories, but admits, however, traumatic influences to a greater extent than Cohnheim does. He even thinks that an injury frequently induces the development of tumors, but only in such parts where supernumerary embryonic cells exist. He says that only in such places containing those supernumerary cells an injury will react, starting a tumor by a "tumor-producing proliferation of cells;" while an injury inflicted upon a part where the cells are normal, will never produce

[1] Epstein, Ueber Epithelialperlen, etc., Zeitsch. für Heilkunde, I., 1880.

[2] Maas: Zur Etiologie der Geschwulste, Berliner Klin. Wochenschr., No. 47, 1880.

that effect. By this hypothesis he tries to explain why thousands of injuries are not followed by the formation of tumors, while in a few instances tumors develop promptly in consequence of a trauma, and this only because in these few instances there must have been present abnormal embryonic cells which gave rise to the development of the tumor.

Hence he thinks that even a traumatic theory of the development of tumors can be established only by Cohnheim's hypothesis.

He quotes several cases where nævi and other congenital formations developed into cancers in consequence of an injury; the latter, however, not being necessary in the presence of the numerous cases of congenital growths on record, and the large quantity of cases of tumors in which a traumatic history cannot be traced.

Hasse[1] in an elaborate paper strongly approves the views held by Cohnheim.

F. W. Zahn[2] claims to have determined experimentally that portions of embryonic tissues engrafted upon animals continued to grow, while all other transplanted tissues of adult animals disappeared sooner or later. (See experiments.) These results, if correct, would be in favor of Cohnheim's theory. Cohnheim's theory is certainly a highly interesting and ingenious one, and not without foundation, though some points are evidently exaggerated. For very able critics of this view see: Samuel,[3] Lang,[4] Osler,[5] McGraw.[6] I am sorry to say, though, that some of these gentlemen did not read Cohnheim carefully, and probably unintentionally misinterpreted and misrepresented his views.

Thiersch and Waldeyer,[7] though not advocating so decidedly the embryonal hypothesis, are yet strongly in favor of it.

Lücke[8] believes in the purely local origin of all tumors, and defines

[1] C. Hasse. Die Beziehungen der Morphologie zur Heilkunde, Leipzig, 1879.

[2] Zahn, Sur le sort des tissus implantes dans l'organisme. Congr. Méd. International, Genève, 1878.

[3] Samuel, Pathologie, l. c.

[4] Lang, Weiner Med. Presse, 1879.

[5] Osler, Canada Med. and Surg. Journal, 1870.

[6] McGraw, T. A. The Germ Theory of the Etiology of Tumors, Detroit Lancet, 1878, p. 803.

[7] Waldeyer, Volkmann's Sammlung Klinischer Vortrage, No. 33.

[8] Lücke, Pitha und Billroth, Handbuch der allgemeinen und spec. Chirurgie, "Geschwülste."

a tumor as a simple multiplication of tissue above physiological limits "It is interesting to remark," he says, "that multiple warts may persist during the whole life of the individual; if, in advanced age, one of the warts should develop into a carcinoma, this transformation will nearly always be limited to such a single wart. Could this be the case, if we take dyscrasia as an etiological basis?"

Lücke says that tumors of all kinds, even the most malignant ones and those which have reached a considerable size before becoming general, may for a long time be latent in the organism without impairing the latter, if the local conditions are otherwise favorable.

Primary multiplicity is peculiar only to benign tumors. But there does not exist a lipomatous or fibromatous dyscrasia.

Lücke admits our great ignorance in regard to the etiology of tumors rather than accept names like dyscrasia, which are only used as a cloak for our ignorance. He, as mentioned before, admits the embryonal theory and believes in an individual predisposition, and to this he thinks should be added hereditary predisposition and general and local circumstantial causes of various kinds, considering, along with the latter, also traumatism.

4. *Idiopathic or Spontaneous Theory*.—Rindfleisch, in his famous text-book on Pathological Anatomy, expressed himself decidedly in favor of a spontaneous origin of tumors. His classical phrase: "Tumors arise spontaneously, but they do not heal spontaneously; while inflammations do not arise spontaneously but they heal spontaneously" —expresses really very perfectly the notion of the practical physician. The persistence, the "organ-like character of tumors," he explains by the fact that tumors follow more the rules of physiological growths. Inflammatory formations, on the other hand, are produced essentially by a conflux of mobile cells at the spot of irritation, hence their rapid appearance and almost traceless disappearance.

Rindfleisch[1] considers the evolution and involution of tissues and organs to be an important factor in the etiology of tumors. Thus, he explains the development of tumors of the connective tissue group by a localized, excessive proliferation of connective tissue elements during evolution (in young persons) and the occurrence of epithelial new growths during involution (in older persons), by a local proliferation of the superabundant epithelial elements. He admits, though, that local irritation plays an important rôle.

[1] Rindfleisch, Handbuch der Gewebelehre, 1875.

Thiersch's hypothesis for the etiology of cancers is, that in advanced age, there occurs in the elements of the skin a disproportion between the epithelium and the connective tissue, the latter losing its resisting power towards the epithelium, which proliferates, responding to a given irritation. Some tissues, he says, become prematurely old. Benign moles and warts may begin to proliferate in advanced age and form malignant tumors.

Dr. Charles B. Nancrede,[1] in his excellent paper, ascribed the cause of the formation of morbid growths to a disproportion in the local vascular supply, in some points in accordance with the views of Thiersch[2] and Rindfleisch. Dr. Nancrede believes that the so-called inheritance of a cancer consists in the relatively earlier atrophy of the connective tissue and the transmission, from father to son, of a peculiar readiness to respond to irritants of the glandular elements. As to the exact nature of the irritation which sets up this abnormal action, he believes that there is, in all cases, some slight mechanical or chemical irritant acting upon the "*locus minoris resistentiæ*," anatomically prepared for the morbid growth, and he defines this irritant not as a violent blow or a cauterizing agent, but anything that increases the amount of blood in the part, however unnoticed or unnoticeable it may be.

In connection with the idiopathic or spontaneous theory due notice must be given to the views of Mr. Payne,[3] who applies Spencer's dynamical laws to the causation or rather to the growth of tumors.

Herbert Spencer says: "Growth is unlimited, or has a definite limit according as the surplus of nutrition over expenditure does or does not progressively decrease. Tumors, having no function, have no expenditure, and hence all the force is used up in growths, and the larger the tumor the more force is liberated and the larger it grows. They are like plants in being almost wholly accumulators; they have no expenditure of force, hence their unrestrained increase in size."

Mr. Payne properly remarks: "New growths are more frequent in passive tissues than in actively functioning tissues. Fatty tissues, bone tissues, and all varieties of growths which originate from connective tissue, are instances of the connection of mechanical passivity with excessive growth." On the other hand, he explains the extraordinary

[1] Nancrede, Transactions of the Pathological Society of Philadelphia, 1876–7.

[2] Thiersch, Der Epithelialkrebs. Leipzig, 1865.

[3] Payne, Origin and Relations of New Growths, Brit. Med. Journal, 1874.

rarity of tumors composed of striated muscular tissue by the strong activity of the latter, the nutritive supply being balanced by the expenditure of force.

S. Stricker,[1] of Vienna, expresses no views of his own on this subject, but it appears that he is inclined to believe in the spontaneous origin of tumors.

5. *Nervous Theory.*—A nervous theory of the etiology of tumors is also more or less ably advocated. I will briefly mention the arguments brought forward :—

Schrœder Van der Kolk[2] after cutting the tibial nerve and breaking the bones of a dog's paw, found that instead of regular callus forming, a tumor grew, resembling a medullary cancer. Hence he ascribes the development of a tumor to want of innervation.

Virchow says, however, that almost the same tumor can be produced by breaking the lower jaw of a rabbit just in the median line where there are no nerves. Many experiments of nerve section with fracture have been tried, but without success.

Lang[3] advocates a purely nervous theory for the etiology of tumors. He argues as follows: "I find that the relation of the nervous system to tumors is very much underrated by some, and by others altogether ignored, yet to me it seems in many cases very probable. . . . .

"There can be no doubt now that lesions or diseases of nerves or their centres result in decided disturbances of nutrition and that these, in several cases, have given rise to increased formations of different kinds: connective tissue hypertrophy, hyperostoses, hypertrophy of the whole side of the face, etc. . . . . In all these cases I place the origin of the diseased condition in the nerve centres, whence it proceeds towards the periphery along the course of the nerve creating disturbance. . . . . Not unimportant appears to me in connection with the observation made lately by Billroth[4] and Esmarch, that malignant tumors are caused to disappear entirely by the use of internal medicine. As the most important remedy to bring about the effect is arsenic, and it is this drug that relieves the most desperate cases of neuralgia.

[1] Stricker, Vorlesungen in der allgemeine und experimentelle Pathologie, II., 1878.

[2] Quoted by Virchow, Krankhafte Geschwülste.

[3] Lang, Wiener Med. Presse, Nos. 16, 18, 20, 1879.

[4] Billroth and Esmarch, Wiener. Med. Wochenschrift, 1871.

"That arsenic affects as an alterative the organism, and brings about a favorable condition in the processes of nutrition, the dermatologist has ample opportunity to observe. Exactly the physiological process by which it acts is yet unknown, but we may suppose that it acts upon the nervous system or that part called trophic nerves which control nutrition. From experiments on animals, we find that arsenic does not enter all organs with the same degree of readiness, but that it enters some much more readily than others. The organs most disposed to take it are the brain and spinal cord.

"Scolosuboff[1] therefore advises, in criminal cases for poisoning by arsenic, to examine the brain, likewise in cases of gradual arsenical poisoning. Hoffman[2] and Ludwig obtained the most pronounced arsenical mirrors from preparations of the brain and liver."

In respect to the possibility of the dependence of the development of tumors upon the central nervous system, Lang makes the following concluding remarks: "It is conceivable and possible to conclude, and by analogy from other processes it is even probable, that if the disturbances of nutrition are controlled by trophic centres, the first impression coming from the periphery, the disturbance of nutrition may exist as the result of a reflex. Regarded in this light we can explain the success of the dietary treatment of cancer; for, Beneke and Esmarch[3] found that when the peripheral terminations of the nerves of the alimentary canal are less irritated, disturbance of nutrition is less likely to occur from reflex action. Individual peculiarities must, however, be taken into consideration, as we know from experience various articles of food have peculiar effects upon the nerve termination of some individuals, and the direct relation of cause and effect can be observed; *e. g.*, where strawberries, lobster, ham, and other articles of food produce urticaria in some persons."

Beneke[4] himself however states, that he does not think that the nervous system is in any way influenced by the dietary treatment in cancer.

Dr. Snow,[5] of London, in his paper on the etiology of cancer, based upon two years' statistics from the cancer hospital, comes to the conclusion that nervous depression, especially mental trouble, is the most

[1] Scolosuboff, Jahresbericht der Chemie, 1875.
[2] Hoffman and Ludwig, Wien. Med. Jahresbücher, 1877.
[3] Beneke and Esmarch, Langenbeck, Archiv, vol. 22.
[4] Beneke, Berliner Klin. Wochenscrift, No. 47, 1880.
[5] Snow, London Lancet, Dec. 1880.

prominent cause of cancers. He says: "Of 103 uterine cases in which preceding causes of mental or physical nervous depression (trouble or very hard work) were sought for, 55 gave a history of previous mental trouble, 15 of hard work alone, while 10 gave doubtful replies. Of 38 breast diseases, 24 patients gave a history of sorrow and anxiety, 4 of hard work, while 1 was doubtful. Thus of the breast cases (although I must confess the number specially investigated in this connection is smaller than I could wish), 73.68 per cent. gave an account of nervous depression, either mental or physical, immediately preceding the appearance of the disease, while in the uterine cases the percentage amounts to 63.1. From this I cannot help inferring that sorrow, anxiety, and hard work are very powerful factors in the production of malignant disease."

After careful inquiry into the history of a large number of cases, he comes to the following conclusions:—

"1. Hereditary tendency, as a predisposing cause of cancer (at all events of mammary and uterine), is almost valueless if not entirely so, and in practical diagnosis should altogether be ignored as misleading.

2. Mechanical injuries directly produce cancer in a certain percentage of cases, but this percentage is small.

3. As direct and immediate causes of cancer (especially in my own experience of uterine cancer), mental trouble and hard work are very potent agents, and exert more influence than any other antecedent within our present knowledge."

II. Experiments.—Positive science rests upon facts as deduced from experiments, and through experiments and the microscope only can the true nature and the etiology of tumors be settled. As yet very little has been done in this line, and the results of experimenters hitherto pertain only to the inoculability or infectiousness of tumors.

On account of a discrepancy among authors as to whether tumors can be inoculated or not, and on account of the different interpretation which they ascribe to the experiments done, I find it necessary to quote all of them, and to show why certain experiments succeeded and others failed.

It seems to me that the main object the experimenters had in view was to find a specific virus in tumors; the nature of the experiments and the results seem to justify such an inference. Furthermore the experiments have an important bearing on the etiology; for this reason,

also, it will be necessary to review them before entering upon a *resumé* of the etiological facts.

Infection certainly may take place in the same individual by metastasis, but whether it takes place from one individual to another is disputed. The products of specific inflammation, viz., gumma, tubercle, lepra, etc., are not included here. There is only one positive statement on record, that of Eiselt,[1] who, in the case of a man suffering from melanotic tumor, asserted that the patient had been infected from horses. In rare cases it has been observed that an ulcerating tumor infects by contact a structure which merely rests or lies upon it.

Lücke had observed the buccal mucous membrane to be infected by contact from an ulcerating cancer of the tongue and to become the seat of an independent cancer. He also directs the attention to the frequent occurrence of cancer in Douglas's pouch, secondary, by direct contact, to cancer of the abdominal viscera.

*a. Experiments: Negative Results.*—The first experiment on inoculation of tumors is carefully recorded by Peyrille in his Latin dissertation in 1774.[2] He injected under the skin of the back of an animal some juice from a cancer of the breast, and thought that on the fiftieth day there was produced a cancer. But from his own description it is probable that the process was rather one of a pyæmic nature, as Dr. Zahn relates.

Dupuytren, Valentin, and Vogel, in their time, also experimented with tumor inoculations upon animals, making injections with "tumor juices or with minutely pulpified tumor particles," but with negative results.

Gohier[3] records that inoculation of horses, asses, and dogs with melanotic tumors have not been successful in his hands.

Virchow[4] mentions that he introduced, without success, into wounds of the skin and the conjunctiva of rabbits and dogs fresh melanotic sarcoma of man.

Billroth did not succeed in inoculating the ears of a rabbit with juice of a melanotic sarcoma from a man.

Hyvertl,[5] desirous of ascertaining the effects of cancer-grafts upon

[1] Prager Vierteljahrschrift, 1861, vol. 70.

[2] Quoted after Zahn, Congrès International Médical, 1877. Genève, 1878.

[3] Gohier, Memoir, p. 338.

[4] Virchow, Kr. Geschwülste, vol. ii. p. 253.

[5] Hyvertl, Gaz. des Hôp., No. 49, 1873. Allg. Med. Centr. Zeit., 1873, p. 602.

the body of rabbits, undertook a series of experiments. The portions of tumor, free from pus, and not far advanced in development, were introduced deep into the tissues as far as the muscular layer. Inflammation and induration resulted, the latter to be felt five months afterward as a "tumor." This was surrounded by a tissue rich in vessels, and presented its previous appearance: cancer-cells and alveoli at the periphery with granular degeneration at the centre; at the latter point was calcification. The autopsy showed that neither infection nor general disease had resulted. The author concludes that young cancer-cells, transplanted from man to animals, do not produce cancer.

Lang,[1] impressed by the idea that heterotopia of cells or tissues gives rise to tumors, undertook a series of experiments, introducing various tissues into different parts of animals; but, in his eagerness to be judicious, he actually destroyed the tissues before introducing them, as can be seen from his own account.

The experiments were made on thirteen dogs, varying in age from a few days to several years. He used either normal tissue or such as had been in an inflammatory condition, introducing it either into the organism of the same dog, from which the tissue was taken, or into other dogs. For this purpose he rendered the tissue to be introduced as fine as possible on a plate, warmed to a temperature of 38° C. He then suspended the pulp in a 12 per cent. solution of carbonate of sodium, of the same temperature, and introduced it into different parts of the animal by means of a hypodermic syringe.

Such experiments Lang carried on with skin, glands of skin, mucous membrane, mucous glands, the nipple of the breast, the mammary gland, cartilage of the ear, joint epiphyses, and with young bone-tissue. All these tissues, prepared as above stated, were introduced subcutaneously twenty-seven times; five times near the bone; twice into the substance of the mammary gland; once into the testicle; forty-three times into the peritoneal cavity; three times into the thoracic cavity; twenty-three times into veins, and eleven times into arteries; in all there were one hundred and sixty-five experiments.

Out of the thirteen dogs, which had been experimented upon, six ran away and some died and seven were under his observation for some time; yet no tumors developed.

[1] Lang, Die Aetiologie der Malignen Geschwülste, Weiner Medicinische Presse, 1879, No. 16.

Zahn[1] transplanted fragments of chondroma into the anterior chamber of the eye, without success.

*b. Experiments: Positive Results.*—Langenbeck[2] injected fresh cancer juice into the jugular vein of animals. Many of them died in from twelve to twenty-four hours, the juice forming so great an obstruction in the lungs that they all died of dyspnœa. One experiment was performed upon a large, strong, two years old dog. He first took from the femoral artery eight ounces of blood, injected a half ounce of cancer juice (taken from a growth two and a half hours after removal), mixed with defibrinated blood, and all was injected into the femoral vein; two days after the operation the dog had a fever and dyspnœa; eight days afterwards the dog was well, but began to get thin; three months later the animal was killed; in the lung was found three flat nodes, which, under the microscope, proved to be cancer.

Virchow, who examined the microscopic preparation, agrees with the diagnosis of Langenbeck, but remarks that it closely resembled congenital cancer of the dog.

Klencke[3] succeeded in inoculating a horse and a dog with a melanotic tumor from another horse.

C. O. Weber[4] injected some cancer juice from a medullary cancer of man into the crural vein of a dog, and also introduced particles of the tumor under the skin; at the end of sixteen days a tumor of the size of a fist was formed beneath the skin, having the structure of a true medullary cancer.

Follin and Lebert[5] injected cancer juice from a mammary carcinoma into the jugular vein of a dog, and in fourteen days cancer-nodes were produced in the heart and liver.

Goujon[6] testifies to having successfully inoculated animals with cancer masses three times in four experiments.

Lebert and Wyes[7] record the following experiments: 1. juice of melanotic sarcoma introduced under the skin of the neck of a rabbit was followed by death in two and a half months; at the point of injec-

[1] Zahn, l. c., p. 633.

[2] Schmidt's Jahrbucher, vol. xxv. 1840, p. 99.

[3] Klencke, Haser's Archiv, 1843, vol. iv.

[4] C. O. Weber, Chir. Erfahrungen und Untersuch., Berlin, 1859, p. 259.

[5] Follin and Lebert, Traité pratique des maladies cancereuses, Paris, 1851, p. 136.

[6] Goujon, Etudes sur quelque points, etc., Thèse, Paris, 1865.

[7] Lebert and Wyes, Virchow's Archiv, xl. pp. 142–532.

tion, in its connective tissue, appeared a pigmented sarcomatous mass, made up of tubers in all two inches square. Internal organs sound.

2. Rabbit injected in neck with juice of melanotic sarcoma; killed in three and a half months. In connective tissue, at point of injection, a pigmented zone, three centimetres long, two centimetres broad. Suppurative pleuritis; but viscera sound.

3. Three rabbits injected with the juice of small round-celled sarcoma of the tibia of a man; no results either local or general.

4. Four rabbits were injected with the juice of human carcinoma; no results.

5. Injected from 60 to 70 grammes of juice of carcinoma of heart into jugular vein of a dog; death in fourteen days. In the heart a number of small nodules of carcinoma and miliary carcinosis of liver.

Dr. Eiselt[1] says that Prof. Klencke, of Braunschweig, inoculated the conjunctiva and lachrymal gland of an old horse, with juice of a melanotic tumor of orbit. In sixteen weeks the entire gland was involved by a similar tumor, causing the eye to protrude. Fragments of same tumor were introduced into jugular vein of a dog, which died in three months, with a melanotic tumor in the left lung.

H. Knapp[2] made inoculations by injections under the skin, and into the crural vein of a rabbit and dog with glioma of the orbit; failure resulted. He succeeded, however, in obtaining increase of bulk of tumor-particles introduced into the vitreous body of the eye, which, while increasing, retained their glioma structure.

The author thinks it possible, however, that the new products (which probably had been adherent to the choroid) may have been of an inflammatory nature.

M. Nowinsky,[3] who experimented in the laboratory of Prof. Wowontzoff, in St. Petersburg, asserts that cancer can be inoculated. He used for inoculation small fragments of a medullary cancer taken from the nose of a dog. Twelve experiments were made on inflamed skin; fifteen experiments were made on normal skin. The first were all negative; of the second two were positive. One of these was as follows:—

On the back of a dog a fresh cut was made and a small piece of can-

[1] Dr. Eiselt, Prager Vierteljahrschrift, Bd. 76, 1862, p. 53.

[2] H. Knapp, Weber Imphungen von Gliom-gewebe vom Menschew auf Kaninchen und Hunde Verhandl. d. natur. his. und med. Ver. Heidelberg, 1868, iv. quoted Medicin. Centralblatt., 1869, p. 79.

[3] M. Nowinsky, Med. Centralblatt, 45, 1876.

cer, 2 c. mm. in size was introduced and the cut sewed up. It healed by first intention; after fourteen days there appeared in the cicatrix a small nodule, of the size of a pea, which seemed to grow very fast, so that eight months after the inoculation, the growth was as large as a walnut. Superficially it had been ulcerated; a month later the dog was killed; the tumor was $3\frac{1}{2}$ cm. in diameter, soft, and on section white. In the subclavicular region a gland was very much swollen. The microscope showed the tumor to be made up of epithelial cells infiltrating the connective tissue. In some places the cells were arranged in alveoli. The lymphatic glands showed metastatic cancer masses. Thus the new formation had the same structure as the tumor, a piece of which was used for inoculation.

In the other case, a piece of the small nodule from the first dog that was inoculated, was put into a young dog three months old, which died one a half months after inoculation from malignant pustule. There was found in the cicatrix a small nodule of the size of a pea; no metastasis. The microscope revealed carcinoma.

A series of experiments of the same character as the foregoing is being carried on at present in the Pathological Laboratory of the University of Pennsylvania by Henry Wile. It would be premature to dwell upon them now, as they are yet in progress. The first series of experiments undertaken by him with injections of cancer juice, failed in every instance. In the second series, so far, of a number of dogs upon which small fragments of carcinoma of man were transplanted subcutaneously, only two have distinct new formations at the seat of the grafts, one of them being of the size of a walnut; the lymphatic glands in the axilla and groin are swollen. The animals will be kept alive until January, 1882. The structure of these new formations is unknown. It was, however, perfectly determined in a third animal (a small dog) that a cancer-graft continued to grow and, retaining the typical structure, reached a tenfold size within two months, when the animal was killed.

In a large number of animals, however, the transplantation failed. The details of the research will be published when completed.

Analyzing the results of the experiments, it is seen that tumors were developed only in those cases where direct transplantation with living fragments of tumors had been made. In no case where tumor-juices were injected were any tumors formed; but at the most, perhaps, only a miliary infiltration of a new growth. Langenbeck's experiments, in

which the injection of juice appears to have been followed by the formation of a tumor, were the only exception to this.

The juices as occurring in malignant tumors are always the products of degeneration of the tissue or cells composing them. The cells suspended in that juice are dead, having undergone fatty or some other degeneration, and this is the reason that injection with juices fails. If particles of living tumor-tissue in which the relation of bloodvessels to cells is preserved happen to be suspended in that juice, "inoculation" might succeed.

*c. Other Transplantation Experiments.*—Paul Bert[1] asserts that any tissue, with the exception, perhaps, of muscle, can be successfully transplanted.

Goujon[2] showed that young marrow of bone can be transplanted with success. He also found that transplantation in very vascular tissues is particularly successful. The experiments were successful only with tissues of the connective-tissue group, and only if transplanted upon animals of the same species whence the graft tissue was taken; otherwise the tissue experimented with was promptly eliminated by inflammatory suppuration or was atrophied, becoming encapsuled in inflammatory connective tissue.

A number of successful experiments of transplantation to the anterior chamber of the eye were made by Dooremal[3] with mucous membrane of the lip; by Goldzicher[4] with that of the nares and the conjunctiva, and with tissue from the Fallopian tube; Schwenniger[5] with roots of hairs. All these experimenters found that the epithelium chiefly proliferated and occasionally formed masses of new formations and cysts. Experiments with corneal and nervous tissue gave negative results. Montegazza inserted various tissues into the peritoneal cavity and beneath the skin. He obtained positive results with bone tissue, testicle, spleen, and intestines, *i. e.*, the tissues grew at the seat of transplantation.

Zielonko[6] transplanted various tissues into the dorsal lymphatic sac of frogs; he succeeded only with corneal tissue, and even here only the deeper, younger cell-layers proliferated.

[1] Paul Bert, quoted after Zahn.
[2] Goujon, l. c.
[3] Van Dooremal, Graefe's Archiv, vol. xix.
[4] Goldzicher, Arch. f. Exper. Pathologie, vol. ii.
[5] Schwenniger, Zeitschr. f. Biologie, vol. xi.
[6] Zielonko, Med. Centralblatt, 1873, No. 56.

Zahn[1] made transplantation experiments on rabbits. Rabbits proved, for these experiments, better than other animals, and the best results attended transplantation to the anterior chamber of the eye and the kidneys. He experimented, however, only with cartilage. With fragments of costal cartilage from adult animals, even when transplanted to animals of the same species, he always failed. He introduced several tissues, fragments of an enchondroma, into the anterior chamber of the eye and into the circulation, but, like other adult cartilage, they invariably underwent fatty degeneration and were absorbed.

Zahn then began to experiment with fœtal cartilage, and always succeeded. The fragments of cartilage continued to grow, though they occasionally calcified in their interior. On one occasion he injected into the circulation cartilage so minutely divided and mixed with amniotic fluid that it could pass through the capillary tube of a hypodermic syringe. At the end of fifty days he found two cartilaginous nodes, one of them of the size of a pea, in the adventitia of a vein. He succeeded also in transplanting fœtal cartilage from one species of animal to another, *e. g.*, from a cat to a rabbit, which shows that the same fœtal tissues of different species of animals are equivalent.

Zahn says that the same results are obtained after experimenting with fœtal bone. He adds: If an entire fœtal organ is transplanted, *e. g.*, a femur, it continues to grow, retaining pretty nearly its form though it accommodates itself to the shape of the organ into which it is introduced. One of these transplanted femurs Prof. Zahn exhibited at the International Medical Congress in Geneva. "Upon its diaphysis there was developed an exostosis, and on its epiphysis some enchondromata," Zahn did not think that his results in the transplantation of tissue deviated much from those of others. Other reliable experimenters succeeded in transplanting only those tissues which contained some young developing or germinating cells or cell layers, such as red marrow of bone, the roots of hairs and periosteum of young animals. Nobody probably succeeded in transplanting nerve and muscular tissue; "the practical surgeon," he says, "will not undertake a resection of bone in an old person in whom the periosteum has lost its

[1] Zahn, Sur le sort des tissus implantés, etc., Congrès Méd. International, Genève, 1878.

osteogenetic properties." Virchow[1] thinks very highly of Zahn's experiments, and considers that his results solve the problem of the transplantation of tissue.

In connection with this it is also interesting to read Ball's[2] monograph, which, however, does not contain anything new.

Cohnheim and Maas[3] undertook some experiments to determine whether a piece of periosteum would grow and develop bone in the lung, being introduced by means of the jugular vein. Experiments were made on rabbits, dogs, and chickens, always taking young subjects. The best results were obtained with chickens. In from three to five days the animals were killed; the embolus could not be detected on the external surface of the lung, but in ten or fifteen days it could be detected as a resisting and hard spot in the parenchyma of the lungs. In experiments, however, of twenty days' duration the results were negative; the emboli had disappeared. In the first three or five days the periosteum thickens and is infiltrated with a large number of wandering cells, sometimes collected in masses; in ten to twelve days hyaline cartilage cells were found; in fifteen to sixteen days bone lamellæ were always found. From these experiments one would be led to believe that small particles torn off from the parent mass were carried off by the bloodvessels, became impacted, proliferated, and in this way a primary tumor gives rise to a secondary tumor. But the following experiments disproved this view: after twenty days the embolus could not be felt in the lung externally; on cutting open the artery the periosteum was found well shrunken together, and of ossification nothing was seen; after the lapse of a month not a trace of the embolus was found. It is just the same in subcutaneous transplantation; it grows for a while but soon becomes reabsorbed and disappears.

Cohnheim and Maas argue now as follows:—

It may be attributed to some physiological condition of the organism which, as in the callus of fracture, throws out of the system all foreign matter. If this is right, then in those individuals who suffer with metastatic tumors, the ability of the organism to resist the introduction of foreign matter is wanting.

At my suggestion a number of experiments were made by Henry Wile, in the Pathological Laboratory of the University of Pennsyl-

[1] Virchow, Archiv, vol. 79, p. 189.

[2] Ball, Das Princip des Wachsthums, Berlin, 1876.

[3] Virchow, Archiv, vol. 70, 1877, Cohnheim and Maas.

vania,[1] to determine whether periosteum transplanted under certain conditions would continue to grow and develop bone, or, as Cohnheim says, disappear at the end of twenty days. With the permission of Mr. Wile, I will briefly give the essential points and the remarkable results of these experiments.

Six of these experiments have been selected and the results are as follows. Dogs were used in every case :—

1. A small piece of periosteum from radius of an adult dog was introduced into the right jugular vein. The dog was killed after thirteen days. In the middle lobe of the right lung near the surface was found a small resisting nodule which on section showed it to be made up of granulation-tissue containing cartilage cells.

2. Periosteum was introduced as before. The dog was killed after twenty days ; a nodule was found in the inferior lobe of the right lung somewhat larger than the preceding nodule. On section, under the microscope it proved to be made up partly of young cartilage and partly of well formed bone.

3. Periosteum from the tibia was introduced into the right jugular vein as before. The dog was killed at the end of thirty-three days ; a

[1] The work of Henry Wile is only one of the numerous experimental researches carried on at present by a number of students on different subjects in the Pathological Laboratory of the University of Pennsylvania. We are extremely satisfied with the excellent facilities for work in the laboratories of this institution, the ample supply of instruments, pathological material and so forth. Original research is liberally encouraged by the faculty and by the chief of the pathological department, Prof. James Tyson. Notwithstanding an endowment fund would be desirable for the purpose of publishing the work which is now being carried on and that which has accumulated during the last few years. So far but very few of the latter are published, and these only in short abstracts and insufficiently illustrated by wood-cuts. The objection to these papers by the editors of American journals is their bulk, the cost of the wood-cuts and plates, and occasionally that the contents are purely of scientific interest. Now these researches should not be published in a crippled manner, and be lost in a host of most wonderful obstetrical and therapeutical curiosities, but should be issued in full, in separate periodical issues and all the best plates reproduced at any cost. In such form they would soon be noticed as worthy products of American histology and pathology, and might be either sent to those members of the medical profession both here and abroad, or would be procured by those who are interested in scientific medicine. At the same time the proper publication of these works would be a further encouragement to students to engage in original research, and thus to elevate their standard and that of their Alma Mater.

nodule was found in the middle lobe of the right lung; this nodule was perceptibly larger than the one in the preceding experiment and was still found to be made up of bone tissue.

4. Periosteum was introduced as in the last experiment. The dog was killed at the end of thirty-seven days. A resisting nodule larger than in the preceding experiment was found in the middle lobe of the right lung; under the microscope it proved to be bone.

5. Periosteum was introduced as before, and the dog killed at the end of fifty days. A nodule was found in the middle lobe of the right lung of the size of a pea; it also proved to be bone.

6. Periosteum was introduced as before. The dog was killed at the end of one hundred days; several hard nodules were found at the periphery of the right lung, one of them being of the size of a large pea, *i. e.*, about one hundred times larger than the original particle introduced. Careful microscopic examination was made; result, bone tissue.

The microscopic diagnosis was confirmed by me in every case.

In each experiment it must be noted, that there was a perceptible increase in size in the periosteal embolus. These facts show positively that the periosteal embolus will not only not disappear at the end of twenty days, but under favorable conditions ossify and continue its growth and development indefinitely.

Hence there must be something wrong in the experiments of Cohnheim and Maas, and consequently all the conclusions drawn from them cannot be substantiated.

[Cohnheim's hypothesis on the etiology of tumors seems of late to have gained some more ground in Germany. While my paper was in press, the just issued (August 8th, 1881) part of Vol. 85, of *Virchow's Archives* reached my hands, which contains an article (*Exper. Nutersuch ueber die Etiologie der Geschwülste*) by Dr. Leopold, who violently supports, and he thinks he has proven by experiments, the view of Cohnheim. I can give here only the mere outlines of the contents of the paper.

The author had made a series of transplantation experiments in the Pathological Institute of Leipzig, under the direct supervision of Cohnheim. Essentially these experiments went only to confirm the experiments of Zahn (l. c.) *e. g.*, that only embryonal (fœtal) tissues transplanted to the anterior chamber of the eye or to the peritoneal cavity will grow; while adult tissues do not grow but become absorbed sooner or later. Successful results Leopold obtained only with embryonal cartilage, transplanting small fragments to the anterior chamber of the eye, where they continued to grow and in about six months reached a bulk three hundred times larger than the original pieces

inserted. Larger pieces of tissues and organs, *i. e.*, the whole head of a fœtal thigh inserted into the peritoneal cavity, did, however, not grow even after the lapse of months, but were peculiarly preserved without decomposing, becoming enveloped in a connective tissue capsule. The fragments of cartilage referred to above had not only considerably increased in size but showed advancing development, viz.: the formation of marrow-cavities and of bone lamellæ.

From this the author concludes, that he has produced experimentally a tumor, an artificial enchondroma.(?) He thinks that he will also be able "to produce artificially in the same manner epithelioma, myoma, adenoma, and dermoids."

I think, however, that he must succeed better with them than with his "artificial enchondroma" which is nothing else than a simple graft of cartilage transforming into bone.

I must confess that Cohnheim's embryonal hypothesis is very seducing, but still it can hold good only for the congenital tumors, viz.: rhabdo-myoma, simple angioma and lymphangioma, dermoids, and perhaps the heterotopic adenomata. For all the rest of the tumors Cohnheim's theory is untenable, as will be shown later.]

The results of all the experiments taken together may be summed up as follows:—

1st. Tumors cannot be inoculated by virtue of a specific poison or of any specific properties.

2d. Fragments of tumors, when transplanted from one individual to another, continue to grow, and may reach considerable size.

III. Statistical Facts.—Allow me now to enumerate briefly the main etiological facts as deduced from statistics and observations, arranging them somewhat after the pattern of Professor Lücke, and to give some additional points, which are of interest in the etiology of tumors.

1. *Hereditary predisposition.*—Broca, in his *Treatise on Tumors*, p. 151, gives some interesting statistics. One of these is as follows:—

1st generation, Madam Z. died, aged 60, from cancer of the breast.

2d generation, four married daughters died, at ages from 43 to 62, of cancer of liver and breast.

3d generation, one of these daughters had seven children.

1st son died young.

2d son died, age 64, cancer of stomach.

1st daughter died, age 35, cancer of breast.

2d, 3d, and 4th daughters died, age 35 to 40, cancer of liver.

5th daughter remained free.

In analyzing the family still further it was established that out of a

family of 26, in three successive generations 15 individuals died of cancer.

According to statistics given by Lebert,[1] the development of cancer as far as hereditary influence appears in a ratio of 1 to 7. Nearly all experienced authorities agree with this.

Other tumors, too, have been traced for several generations, either upon the same or upon different parts of the body.

Dr. Snow, of the London Cancer Hospital, Birket and Wilks, of London, and some others, consider cancer not to be hereditary.

2. *Age* predisposes to tumors and certain ages to special forms of tumors. According to Virchow, the frequency of the occurrence of tumors in general stands in direct ratio to the age up to the 71st year.

In reviewing the predisposing and exciting causes of mammary neoplasms, S. W. Gross[2] arrives at the conclusion "that their development is connected with the changed proportions of the component tissues of the breast at different periods of life, and that the condition of the tissues is, as a rule, indicated by the age of the patient." He agrees with the opinion that the evolution and involution of tissues, as conditioned by age, plays an important rôle in the etiology of tumors. A "prematurely old breast," Dr. Gross says, is prediposed to carcinoma.

3. *Sex* predisposes to tumors. Statistics given by various distinguished writers show that neoplasms in general are more common in the *male* than in the *female*, with an averaging ratio of 6 to 4.

This ratio differs somewhat in the case of cancers.

4. *Certain physiological periods of life* predispose to tumors—*e. g.*, adenoma—dermoid cysts.

Statistics show that marriage and the menopause predispose women to tumors.

5. *Social conditions*, prosperity, and good life predispose to tumors.

6. *Certain kinds of food* are supposed by some to promote the development of tumors, especially of malignant ones. It is said that predominately vegetable food predisposes to cancer.

Roll,[3] giving an interesting comparative study of tumors in animals, states, however, that cancer is more common in the dog than in the herbiverous animals.

[1] Lebert, Traité pratique des maladies cancereuses, Paris, 1851.

[2] Gross, Tumors of the Mammary Gland, Philada. 1880.

[3] Lehrbuch der Path. und Therap. der Hausthiere, Wien, 1860, p. 257.

In the western portion of the United States there is a popular belief that tomatoes produce cancer. So much importance was attached to this belief that the question was made the subject of an investigation by the Michigan State Board of Health, which, however, failed to establish any ground for this opinion.[1]

7. *Race.*—It would be interesting to know whether certain races and nationalities exhibit any special predisposition to the development of tumors.

It is generally supposed, Lücke says, that people of the south are less predisposed than those of the north. It may be so for cancer and sarcoma, but it is well known that connective tissue growths (elephantiasis) and lipoma, in Hottentots, are found very frequently in the south. In negroes, tumors of the skin are very common.

During my visits to Egypt in 1868 and 1872, I was struck by the frequency of tumors among the native Arabs! Sometimes they were of considerable size and mostly, I think, located about the neck, face, axilla, and lips, or multiple tumors in the skin.

Their nature I do not know, as I was not interested in medicine at the time. I failed to discover any records of tumors in those countries, except those which I will quote.

Dr. Fraue,[2] who travelled for many years in Asia Minor, Syria, Palestine, Mesopotamia, Egypt, and Nubia, found that in these regions cancer especially and also epithelioma of the lip was almost unknown;(?) also Dr. Heinrich Bartle, the famous African traveller, who made observations in these regions for a period of five years, states that he saw only one case of cancer and that on a man. Only in one race of Barbars and Arabians near the river Niger did he find an affection of

[1] This belief has been traced by Dr. Wyman to the following source: J. D. Hylton, M.D., Professor of an Eclectic Medical School, writes as follows in the *Eclectic Med. Journal of Penna.*, Sept. and Oct. 1871. I will quote here the article as a curiosity: "In my chemical researches in the vegetable kingdom I am daily discovering new agents. In the tomato I can isolate a pure alkaloid, which possesses cholagogue properties superior to podophyllin, leptandria, euonymus, mild and efficient in its action. But in my microscopical researches with this same agent I have discovered *cells identical with cancer cells, the appearance of the two, the true cancer cell and those found in the tomato, being under the microscope the same.*" The learned quack adds: "If this fact is true and can be substantiated by other chemists, it may in some manner account for the fearful increase of cancer in sections of the country where this fruit is cultivated." (!!!!)

[2] Virchow's Archives, vol. 25, p. 602.

the skin which resembled very much epithelioma of the lip. Livingstone and Lantre did not meet cancer in any of the African races. Lantre, who spent many years among the Bassuti, a nation north of Cape Colony, did not see one case of cancer of the face, which among the whites of the Cape Colony is not at all rare. Epithelioma of the lip is most frequent in Central Europe, which is indeed the most thoroughly known region.

Dr. M. Grau, of Detroit,[1] inquired particularly about the prevalence of tumors among the Sioux, Bannocks, and other Indian tribes (with whom General Custer came in conflict at the time of his death), of Dr. Gillycuddy, who for three years past had been acting as assistant surgeon to a regiment of United States cavalry, stationed in that region. The doctor had come in contact with many thousand Indians, and was in medical charge of one of the largest Indian agencies. In an extensive practice among them, in which he had occasion to treat them for almost everything else, he had met with but one tumor of any kind, and that was an atheromatous cyst on the neck of a squaw.

I would like to quote at this place some remarks of Dr. Crisp, of London, made on occasion of a discussion on cancer.[2]

"The geographical distribution of the disease, Mr. Haviland has shown, in the cancer chart which he published, corrected from the records of the tables of the Registrar-General, that there are cancer-fields in England where cancer predominates to an inordinate extent, and that these cancer-fields are associated with certain local peculiarities of soil, geological peculiarities. It is well known, no doubt, to many members of the society that, in those districts where the primitive rocks and the earlier formations principally exist, cancer is comparatively seldom met with; whereas, as you descend the river courses, and get into the tertiary formations, and especially into low-lying alluvial lands subject to overflowing, and other conditions of that kind, you find cancers developed to a much greater extent. The subject is as yet not worked out sufficiently."

"In country places the people constantly marry within a circle. One individual who happens to have a cancerous taint may produce five or six cases of cancer, as they seldom go far from the locality. The prevalence of cancer may be more readily accounted for in that way than by any other influence that locality or soil may exert."

[1] M. Grau, The Germ Theory of the Etiology of Tumors, Detroit Lancet, 1878, p. 803.

[2] British Medical Journal, 1874.

8. *For endemic* conditions influencing the etiology of tumors, goitre if regarded as a neoplasm would be the only instance.

9. *Acquired predisposition* to tumors. By this I would like to define a predisposition acquired through *external influences*, *i. e.*, through anything that may excite an inflammation or a long continued irritation and consequent disturbance in the tissues; *e. g.*, injuries, long standing, pressure or irritation, cold, etc. Injuries are properly regarded as exciting causes of tumors, but this may only be so in a certain class of cases, perhaps in hereditary tumors. From my inquiries I am inclined to believe that the inflammatory process creates conditions in the tissues which directly and more than any other cause predispose to tumor formation, and hence I would regard inflammation a predisposing, rather than an exciting cause. Good and exhaustive statistics should be made in this direction.

Of all the enumerated causative factors I will give a detailed consideration only to the last one, the "*Acquired predisposition*," suggested by myself. The rest of the supposed factors are all, to my mind, not sufficiently established yet; though some of them, *e. g.*, "the evolution and involution of tissues" as conditioned by age, are of great importance in the etiology of tumors.

IV. Personal Investigation and Arguments in favor of the Inflammatory Theory.—The immediate cause of tumors has been repeatedly traced, beyond doubt, directly to inflammatory processes. Hence an inflammatory theory of the etiology of tumors is not an hypothesis.

The dyscrasia, embryonal, spontaneous and nervous theories, as ingenious as they are, can hardly, at present, be regarded as theories, but must be called hypotheses; for so far, not a single tumor can be proven to have really developed from the causes promulgated. I gave those hypotheses due consideration, but I did not attempt to criticize them for want of time and space; and, again, there is *nothing to be disproven where nothing is proved.*

I will now enter into some details of the inflammatory view, the one I have adopted.

It is properly held by some that no line of distinction can be drawn between true tumors and chronic inflammatory products. I shall bring forward some facts now generally acknowledged, and also some investigations of my own, which will considerably strengthen this view.

Practically we do not know what inflammation really is; we know only some of its causes, symptoms, and some of the terminations. But the inflammatory process itself and some of its terminations have been pretty well studied and are well known through the labors of Virchow, Cohnheim, Stricker, Ziegler, Ranvier, and Samuel abroad, and W. F. Norris, Woodward, and E. O. Shakespeare in this country. From these observations we also learn that many, and sometimes all the signs of inflammation may be wanting in that process, and really, the symptoms are altogether absent in many of the so-called chronic inflammations.

Many of the products of inflammation are not only difficult to distinguish from tumors, but are actually recognized as true tumors. The criterion of true tumors is regarded to be their tendency for permanency in contradistinction to inflammatory products which tend to disappear; but it can be shown that, while true tumors occasionally do disappear, inflammatory products, very frequently, never disappear.

There are many cases of sudden and gradual disappearance of tumors on record. I shall mention only a few.

Dr. Th. Dwight[1] reports a case of an unmistakable tumor of the rectum, which had disappeared spontaneously. In the discussion of the paper the argument was brought forward that, of all tumors, only lymphoma is known to disappear and, as this tumor was single, it probably was not lymphoma, the latter tumor always occurring as a multiple growth; the probability being more in favor of its having been medullary sarcoma (commonly mistaken for encephaloid in former times).

Prof. Louis A. Duhring[2] has met in his practice with a peculiar variety of tumor, which he has called an *inflammatory fungoid neoplasm*. These appeared suddenly as round or oval, circumscribed, nodular or fungoid growths of a dark-red color, from the size of a pea to that of an egg. Having attained a definite size, as a rule, these growths would soften, diminish in volume and undergo sooner or later spontaneous involution without pigmentation and without scar. Although resembling sarcoma, as Dr. Heitzmann of New York pointed out, Dr. Duhring considers that this disease is unquestionably an inflammatory new formation, allied to sarcoma, but differing from it as

[1] Dr. Th. Dwight, The Disappearance of Tumors, Boston, Med. and Surg. Journal, 1880, p. 562.

[2] Duhring. See Supplement to a case of Inflammatory Fungoid Neoplasm, by Louis A. Duhring, Philadelphia, 1880, pp. 12–16–18.

described by authorities. The great peculiarity of these growths was their rapid rise and fall, rising in a night and disappearing within a week.

H. Fisher[1] records a case of a man from whom a large tumor of the neck had been extirpated; two days after the operation a swelling of a gland, as large as a fist, disappeared during a night; at the same time high fever set in and the patient died. Three days later at the post-mortem no cause of death could be found. In the axilla was found a small swollen gland. Fisher believed that a very acute fatty degeneration and reabsorption of the tumor elements took place. In a second case a tumor of a lymphatic gland as large as a fist diminished to the size of a small apple during the progress of an acute meningitis and tubercular pericarditis. In a third case during ilio-typhus in a girl sixteen years old, a lymphatic tumor of the neck, 5 cm. long and 3 cm. wide, diminished to the size of a bean. In a fourth case a goitre disappeared during the progress of scarlatina. The author adds a few cases of tumors in which, after trifling interference, noticeable diminution of size occurred.

Berns, of Amsterdam, also records similar instances of the disappearance of tumors.

Lücke[2] also observed tumors diminish and even permanently atrophy under the influence of exhausting diseases.

Virchow[3] says that warts, condylomata, and even fibromata have frequently been observed to heal and to disappear, undergoing a slow atrophy and resorption.

Simon[4] records the case of a recurrent fibroid which disappeared completely when treated by cold.(?)

Prof. Wm. Goodell[5] says that fibroid tumors, when affecting the womb at a period near the menopause, frequently undergo retrograde change. "The climacteric once reached, these growths generally grow smaller and may even disappear."

Even Rindfleisch, in his text-book, records that pedunculated tumors have fallen of their own accord, and that entire cancerous nodes have been cast off spontaneously.

[1] Deutsche Zeitsch. f. Chirurgie, xii. Heft 1 und 2, 1879.

[2] Lücke, l. c. p. 16.

[3] Virchow, Geschwülste (l. c. p. 359).

[4] Simon, Discussion on Cancer, Brit. Med. Jour., 1874.

[5] Goodell, Clinical Observations on the Radical Treatment of Fibroid Tumors of the Womb. Transactions Med. Society State of Penna., 1880.

Drs. Ripley[1] and Robinson, of New York, each recorded a case of complete disappearance of an epithelioma. Dr. Robinson's case was one of epithelioma of the larynx which had perfectly healed. Dr. Ripley's case is particularly interesting, as it formed the subject for discussion in the Pathological Society of New York. The case was an epithelioma of the lip of several years' duration, which was not removed on account of the bad health of the patient. Subsequently the new growth spread by extension to both submaxillary glands. While the secondary deposits below the chin continued to grow, producing tissue destruction, the original growth on the lip healed and was fully replaced by perfectly healthy scar-tissue. In the discussion the following points of interest were brought forward by Dr. Satterthwaite and others: Cancers occasionally heal spontaneously, or a cure is accomplished through an acute inflammatory process induced by means of local applications: the healing being a process of cicatrization. The connective tissue proliferates, "squeezing all the cellular elements (of the cancer) to death," and forms dense scar-tissue.

I would like to remark here, that the healing process of an ordinary granulating ulcer is precisely the same as in cancer, wherever healing of the latter occurs. In both instances healing is accomplished by the additional formation of connective tissue, *i. e.*, cicatrization. Poultices, pressure, etc., act beneficially in the healing of ulcers, only because they promote the transformation of granulations into scar-tissue and induce a fatty degeneration of superfluous cell masses; they assist the connective tissue in "squeezing to death" the exuberant granulations, from which to sarcoma there is only one step. Lücke[2] says that sarcomata in very young individuals, occasionally grow as rapidly as acute abscesses and are frequently mistaken for the latter.

I mention this in order to point out the close analogy that exists in the termination of tumors and that of inflammatory products.

We have seen that tumors occasionally heal and disappear. On the other hand, it is well known that many inflammatory products, particularly chronic ones, never do disappear, and that the symptoms and cause of them are frequently less obvious than in the case of tumors. The connective tissue which, in proliferating, constitutes the main

[1] Ripley, Epithelioma of Lip—Spontaneous Healing of the Original Lesion, N. Y. Medical Record, July 16th, 1881. This is only one of the several cases which I have seen recorded since the reading of this paper.

[2] Lücke, Die allg. chir. Diagnostik d. Geschwülste, Volkmann's Klin. Vorträge, 97, 1875.

bulk of elephantiasis and of the cirrhosis of organs and a good many other pathological tissues outside of tumors, never disappears.

Virchow properly considers elephantiasis Arabum and soft fibroma morphologically and etiologically identical and, in the same sense, he does not admit any difference between the connective tissue of an advanced cirrhosis of organs and that of a diffused fibroma. In fact, we are only in the habit of calling a proliferation of connective tissue in the mamma an inter-canalicular fibroma, because the connective tissue affects an external part, while a similar affection of the liver or kidney we term an inflammatory one—a cirrhosis. Why should we make such a distinction?

I believe that inflammation is the most prominent cause of tumors. I am also ready to assert that inflammation is not only an exciting, but mainly a predisposing cause of tumors.

Any inflammatory process, due either to external or internal injury or irritation, etc., may produce a new formation of tissue—a tumor. This may depend particularly upon an imperfect process of healing, as I shall show later.

We even do not need here to take into consideration gummata, tubercle, lupus, etc., the well-established products of inflammation, which so frequently occur as well-defined tumors. I think we can come to a satisfactory conclusion on the inflammatory origin of the true neoplasms even without them.

My own experience is limited, but in the cases of tumors, in which I have had the opportunity to get the history myself, or where I insisted upon an exhaustive anamnesis in cases of others, it was possible in nearly one-half of the cases to trace out a local inflammatory process preceding the tumors at some time or other. Sometimes it dated years back. Careful inquiries nearly always revealed some cause, viz., an injury, long standing irritation, mechanical or toxic, or an impaired or excessive use of the part, pressure, or a long standing catarrh, or something of that nature.

Tissues which are most liable to be the seat of inflammation are also the most *common seat* of tumors. Again, those tissues which do not participate in active inflammatory processes (ganglionic and striated muscular tissue) seldom or never give rise to tumors.

The extensive and careful statistics of Dr. d'Espine, of Geneva, show that the os uteri and the stomach are the most frequent seats of primary cancer, and they are also distinguished for their remarkable liability to catarrhs. Virchow has repeatedly pointed out in a catar-

rhally inflamed gastric mucous membrane the gradual transition to carcinoma.

Dr. J. H. Musser directed my attention to the fact that primary cancer of the gall-bladder is nearly always preceded by gall-stones. He exhibited a beautiful specimen of recently developed cancer of the gall-bladder to the Pathological Society of Philadelphia, in which the clinical history revealed gall-stone for years. In looking up the literature, Dr. Musser found numerous cases of primary cancer of the gall-bladder, and every one was preceded and accompanied by gall-stones. Unquestionably in all these cases the stones excited a catarrhal inflammation and this produced the cancer. A gradual transition from catarrhal inflammation of the mucous surface of the gall-bladder and duct to cancerous formation was distinctly demonstrated in the microscopic preparations from Dr. Musser's case.

I have on several occasions contributed[1] to prove, that most of the so-called indolent ulcers are epitheliomata; nearly all those everlasting ulcers are surface cancers. But at one time they were little sores and were produced by an injury. There are numbers of these indolent ulcers in Philadelphia hospitals, they are all due to inflammation, which is directly traceable. I examined many of them microscopically, and nearly every one proved to be an epithelioma.

Dr. S. W. Gross[2] is of the opinion that cancer of the breast may result from ordinary eczema or psoriasis of the nipple, just as epithelioma of the tongue may follow ichthyosis or hyperplasia of the epithelium of that organ. Dr. Gross finds from his own statistics, that non-carcinomatous tumors, too, have been traced to a trauma in one example out of every eight and a half cases.

Dr. A. P. Gerster[3] presented recently to the Pathological Society of New York three specimens, which illustrated beautifully the traumatic origin of cancer. The first case was a cancer of the sole of the foot which had killed the patient by metastasis of the growth to the brain and nearly all the other inner organs. The doctor had observed the case for years, and had traced with absolute positiveness the primary tumor of the foot to a simple erosion of the skin from stepping on a nail. The second case was cancer of the outer malleolus, also directly formed at the seat of an injury. The third case was a cancer

[1] See Transactions of Pathological Society of Philadelphia.

[2] Gross, Tumors of Mammary Gland, 1880.

[3] Gerster, Specimen illustrating the Traumatic Origin of Cancer. N. Y. Medical Record, July 16th, 1881.

of the lower extremity, developed directly in a scar, the result of a burn dating thirty years back. The tumor did, however, not develop until a year prior to the amputation, when he had struck himself accidentally upon the same spot.

I have seen, myself, several similar cases of tumors positively of traumatic origin, in the University Hospital Clinic and elsewhere. As some of them are and others will be recorded in the proceedings of our Pathological Society, I will refrain from mentioning them here individually.

Winkel,[1] who investigated exhaustively the etiology of myomata of the uterus, came to the conclusion that these tumors are caused either by direct excitents, viz.: coition, injury, abortion, rough removal of placenta, cellulitis; or indirectly: through repeated lifting, shock, sudden hyperæmia, etc. "These," he says, "inevitably first produce disturbance of circulation, stasis, and wandering out of white blood corpuscles, etc." What do we need more; is it here not plain that the inflammatory process was the causative factor of the new growth? The author, however, unnecessarily adds: "this extravasate or transudate gives the impulse for the new-formation like an ovulum, etc."

Epithelioma of the lip, one of the most common tumors, gives a clinical proof of the inflammatory theory; here the irritation by the tobacco juice, as well as the pressure of the pipe, must be the cause, as the new growth occurs pre-eminently in men who are inveterate smokers.[2] I examined the teeth in three cases of epithelioma of the tongue; in every case they were bad, many being broken, and had been in that condition for years; probably the irritation and injury to the tongue, produced by their irregular points, were the cause of the new formation. Similar observations have been made by others.

Epithelioma of the penis has repeatedly been traced to a congenital or acquired phimosis, a condition which naturally occasions constant irritation which usually results in repeated attacks of inflammation.

The workmen in coal-tar and paraffine manufactories suffer very frequently from acute and chronic inflammations of the skin. Volkmann[3] has already described several cases in which true epithelial

[1] Winkel, Volkmann's Samml. Klin. Vorträge, No. 98.

[2] I once saw an epithelioma of the lip in an Irish woman; upon inquiry I learned, however, that she had indulged in smoking a short pipe for many years.

[3] Volkmann, Sammlung Klin. Vorträge.

cancer was developed from those chronic inflammations, and Tellman[1] now adds another of the same nature, ending fatally after numerous operations. This form of cancer has a parallel in the chimney-sweeper's cancer.

Most of the myelinic neuromata occur only in amputated stumps, developing at the cut ends of nerves, and hence are direct inflammatory products. (Perls.)

Frequently warts, nævi, and keloids,[2] through interference which sets up an inflammation, increase and multiply, and are even converted into malignant tumors.

Lücke and Virchow found that whenever an autopsy revealed cancer or any tumor of stomach or œsophagus, the clinical history nearly always revealed "*drunkard.*" We have seen before that from long-continued catarrh to carcinoma there is only one step.

Lipomata very frequently occur in portions of the body which are subject to excessive pressure or irritation. Probably, however, we must first have the development of connective tissue, a fibroma, before we have a lipoma.

Extremely frequent is the occurrence of sarcoma in young persons in consequence of direct injury, or developing in any imperfectly healed scars. Hundreds of cases of chondroma and osteoma, too, have been traced by a distinct and clear history and evidence directly to blows, fractures, cuts, and other injuries.

Any one can convince himself of the above-mentioned facts by looking carefully over the literature of the subject, and by taking exhaustive histories of his own cases. Hundreds of tumor cases of positively traumatic origin are also recorded in the classical works on tumors by Virchow, Weber, Müller, and Broca. All the present younger working pathologists in Europe are in favor of an inflammatory origin of tumors, though none of them expresses himself definitely, still they return gradually to the view which the fathers of Pathology held originally.

Inflammation is the only factor which has been traced to be the positive cause of tumors in a number of cases. This is proven by high authority and statistics. But as these authenticated cases of inflam-

[1] Tellman, Deutsche Zeitschr. für Chir., vol. xii.

[2] Concerning keloids I would like to remark that, as is well known, most of them consist morphologically of cicatricial tissue. Surgeons who remove them find that they always return. They do not return, but the *scar tissue* returns —as in loss of substance true skin is never reproduced, but only a scar.

matory origin are in moderate number, and as those with no cause, by reason of careless note-taking, are in an enormous majority, the inference is drawn that inflammation has little or no significance in the pathogenesis of tumors.

I beg leave to argue as follows: In a certain number of cases it is positively known that inflammation preceded and was the cause of the new growth. In regard to the remaining cases of tumors we know nothing, no positive cause could be traced. Hence I think it logical, for the present, to consider inflammation as the cause of tumors in general.[1] All other alleged causes are only speculations; and nothing reasonable can be brought forward against the inflammatory theory. Speculations are valueless, I think, in the presence of positive facts, even if these be few in number. In science any amount of negative results are always disregarded in the presence of even a few positive facts. *Until contrary proof be given we are at present, by a mass of evidence, forced to the conclusion, that tumors represent merely one of the terminations of inflammation.*

The question now arises in what way does inflammation produce a tumor, and why and when does a tumor develop after an injury? Why is not every injury followed by a tumor if inflammation is the cause? Prof. Maas's[2] ingenious answer was, that it depends upon the presence or absence of Cohnheim's supernumerary embryonic cells at the seat of the injury. If those misplaced or aberrant cells happen to be present in a part, a trauma will induce inflammation followed by a tumor; if no extra cells are present, a simple inflammation will follow and nothing more. But this is only a hypothesis, it cannot be demonstrated. Embryonal (fœtal) cells could not continue to exist unchanged in the adult individual; nor do they need to be pre-existing in order to form a tumor. They can be and are always created by any inflammatory process.

I will try to answer the above question by facts, which microscopic examination reveals and which will show that the study of histogenesis must go hand in hand with that of the etiology, and possibly might disclose the mysteries of the causes of tumors.

It is true that not always direct observation of active pathological

[1] I would exclude here only the purely congenital new formations, *e. g.*, simple angioma, and lymphangioma, rhabdo-myoma, the dermoid cysts and a few of the nævi. These are simple congenital anomalies of the organism.

[2] Maas, Berliner Klin. Wochenschrift, No. 47, 1880.

processes can be made. In the case of tumors only inferences of previous cell activity can be drawn from the microscopic picture; but the pathological process can frequently be traced out under the microscope, from the various transitional stages of the elements of the new forming or formed tissue.

It is in accordance with the modern views to say that every tumor has its strict physiological prototype. Even for the cancer, only the peculiar atypical arrangement of the cells remains a criterion, while the cells themselves are supposed to be strictly identical with those found normally.

It appears to me, and the more I study the histology of tumors the more I become convinced, that any variety of cells composing a tumor are not identical with those found normally but resemble those met with in chronic inflammatory products.

In tumors, the shape and the peculiar variations in size of the cells and nuclei, the character of the intracellular network, and of the amœboid motion of certain cells, the intercellular substance, the occasional arrangement into nodes, the relation to reticulum and blood-vessels, and the peculiarity of the latter are all precisely like what is found in chronic inflammatory products and not like that in normal tissues.

There is a great difference between the tissue elements of fibroma and those of normal connective tissue, for example.

I shall give briefly the details of my investigation of the structure of fibroma which, when completed, will be published and illustrated elsewhere.

Concerning the structure of normal connective tissue, the following seems to be generally established and in good preparations quite demonstrable:—

The ultimate connective tissue fibrils (the fibrillar variety) are in varying number united together to form bundles; these again occasionally unite to form larger bundles; these bundles arrange themselves at different localities in various manners, *i. e.*, parallel as in tendons, or as a lattice-work in membranes, or decussate at different angles and in all possible directions in all other localities, leaving between small spaces. These spaces being dependent for their shape and size upon the arrangement of the bundles. They communicate with one another and thus form a system of channels throughout the whole connective-tissue system of the body. These channels contain a small amount of fluid containing *Mucin*, and they are the receptaculi

of the sometimes enormous quantities of serum in œdema. These same spaces or channels may also get filled with air producing emphysema beneath the skin and in other parts of the body.[1]

V. Recklinghausen has shown that the spaces in the connective tissue communicate with the lymphatics, and he calls the spaces "juice channels;" they act as "vasa serosa" (Orth), conducting the serum from bloodvessels to the lymphatics, and "feeding" (Tyson) the tissues.

By the nitrate of silver method of V. Recklinghausen, which is now the common property of all the laboratories of the world, it can be easily demonstrated that each of the connective tissue bundles spoken of is surrounded by a distinct membrane composed of large flat cells. These flat, so-called endothelial, cells are very thin, nucleated, and are closely united at their periphery with one another, so as to form continuous membranes or sheaths, which envelop each or several fibrillar bundles, and thus at the same time form a lining for the spaces between them. Without nitrate of silver the endothelial cells cannot be seen; all that is seen are the nuclei of the cells: round or oval in shape if viewed from above, or spindle-shaped if the whole cell is seen in profile. I will not enter into further details here; this suffices to make myself now intelligible concerning some points in the histology of connective tissue tumors, particularly fibroma.

I investigated by the nitrate of silver method three specimens of fibroma: 1st, a small, hard fibroma from the finger of a girl, æt. 20, developed from the tendon; 2d, one of the size of two fists from the broad ligament of a woman, æt. 35, and 3d, an intra-uterine fibroma of the size of one fist from a woman, æt. 40.

I might say at the outset that, in the preparation of the first and third specimens, I failed altogether to discover any perfect endothelial sheaths surrounding the bundles of fibres, which were so beautifully seen in a preparation of tendon, made for comparison simultaneously with the fibroma specimens. In specimen No. 2 only a few perfect endothelial sheaths were visible. The microscopic picture of one of the silver preparations (from specimen No. 1) was this: The fibrils were

[1] The subcutaneous tissue of the whole body can be filled with air so as to produce enormous emphysematous disfiguration, by forcing air through blowtubes at a few points or possibly even from only one point of the body below the skin. I have seen children purposely prepared in this way for mendicant purposes.

on the average much thicker than in normal connective tissue; some running straight, others rather wavy and not quite parallel with one another, frequently decussating. Only a few perfect fasciculi or bundles of fibres were seen, but most of them had not a trace of endothelial ensheathment. Some had a partial endothelial sheath in some places, and here the bundles appeared constricted. In several places were seen irregular protoplasmic masses apparently in connection with the fasciculi, which proved to be partially detached endothelial cells. Between the bundles were seen groups of young indifferent cells, resembling white blood corpuscles. Other cells were double the size of the latter, some spindle-shaped and with prominent nuclei. The latter were seen occasionally in a state of division or were already divided. They resembled remarkably the germinating endothelial cells from serous surfaces, as described by E. Klein, of London, represented by him in his *Atlas of Histology*, plate vi.

I interpret the microscopic picture as a whole, thus: the endothelial cells composing the sheaths of bundles of connective tissue have become isolated, and hence the sheaths are destroyed. The boundaries being removed, the liberated connective tissue elements grow with great vigor. The growth is perhaps still more promoted by the presence of the serum of the juice channels, with which the cellular and fibrillar elements now come in direct contact, the sheaths being destroyed. The cells and fibres here, as in elephantiasis, "feed" (as Prof. Tyson would say) upon that serum in which they are soaking. The endothelial is proliferating (germinating, Klein), and probably gives rise to those groups of indifferent cells which evidently form the main source of the new growth. Foerster[1] has pointed out that, in the development of fibroma, the fibres arrange themselves more or less concentrically around, and develop from these islands of cells, thus giving rise to the lobulated appearance of this new growth. It is also very probable that emigrated white-blood corpuscles assist in forming those collections of cells.

What interests us at present, however, is the absence of the endothelial sheaths of the connective-tissue bundles in the fibroma, and that fibroma has this feature in common with all connective-tissue formations, which owe their origin to inflammation, as will be shown directly.

I can affirm the absence of endothelial sheaths in the new-formed

[1] Foerster, Atlas der mikroskopischen und pathologischen Anatomie, Leipzig, 1855.

fibrillar connective tissue as met with in the cirrhosis of organs which invariably accompanies the proliferation of the alveolar connective tissue in such situations. It would be very desirable that other histologists should undertake research in this direction.

Cornil and Ranvier[1] describe the disappearance of the endothelial ensheathments in connective tissue which is the seat of inflammation. They describe the appearance as follows: "The fasciculi are smaller; less distinctly fibrillar; they do not appear to be enveloped by a special layer which limits them, and which causes them to swell irregularly when acted upon by acetic acid." C. and R. consider that the "large flat cells" are replaced by embryonic tissue.

The inflammatory process is, to my knowledge, the only factor which can disconnect or isolate endothelial or epithelial cells united together to form a certain lining or covering. Let us take, as an instance, the lung. The flat cells which form the lining of the air-vesicles are so closely united or grown together in the normal adult individual, that no means at our command can at present isolate them. But in catarrhal pneumonia the inflammatory process demolishes that lining instantly, the cells which compose it "return to their embryonic state" (Stricker), they become completely isolated.

The abnormal increase in bulk of tissue in both the fibroma and the inflammatory connective tissue products, appear to me to be due to the same cause:—

1. The removal of the boundaries which keep the fibres intact, viz., the destruction of the endothelial ensheathments.

2. The proliferation of the endothelial cells of these destroyed sheaths and of the connective tissue elements themselves, probably with the aid of white blood corpuscles.

If the endothelial sheaths of the connective tissue bundles and other normal boundaries are re-established in the inflamed tissue, then it will return to its normal state, or in case of loss of substance will heal by permanent scar-tissue. The healing process is then perfect.

On the other hand, the same tissue will give rise to a fibroma if this healing process is imperfect, *i. e.*, the endothelial ensheathments are not re-established, the connective tissue elements remaining freed from any restriction, proliferate on their own accord, grow above the physiological limit, and thus inflammation terminates in a tumor.

Hence, upon histogenetic grounds, I would suggest: that *fibroma*

[1] Cornil and Ranvier, a Manual of Pathological Histology, translated by Shakespeare and Simes. Philadelphia, 1880.

*should be classed as a product, or rather as one of the terminations of inflammation.*

This is also in accord with clinical experience.

Now, is an inflammatory origin less evident in other tumors? Can there be shown any positive microscopic difference, for instance, between a mass of inflammatory granulation-tissue and a sarcoma? It cannot. To my knowledge distinguished histologists have repeatedly had sad experience of this.

If the discoveries of Classen and Woodward should prove correct, we would, to my mind, have another additional proof that cancer is only one of the terminations of inflammations. I will quote the following:—

Woodward[1] says: "My own studies of thin sections lead me to the conclusion that the migration of white blood corpuscles played a great rôle in the development of cancerous growths, and that at least in certain cases the cancer-cylinders were formed by the transformation of these corpuscles, which first accumulated in the lymphatic capillaries and the passages leading to them."

Classen[2] is even still more positive, saying that he has proven "that the cells of cancer-cylinders and all the elements of cancerous growths are no other than migrated white blood corpuscles escaped from the bloodvessels."

Though in my own researches I have not succeeded as yet in confirming the observations of Woodward and Classen, they are possibly correct, and I utilize them as coming from such high authority. Besides, they correspond so remarkably with what I believe I have established for fibroma.

My view of the histogenesis of fibroma holds good also for primary glandular carcinoma.

The glandelemma or basement membrane in glands (wherever such exists), upon which the epithelial cells rest, may be destroyed in precisely the same manner as the endothelial sheaths of the fibrillar bundles. This is demonstrable in carcinoma beginning to develop in a gland, or in the transformation of an adenoma into cancer. Here as in fibroma only an inflammatory process can accomplish this destruction of the normal boundaries. These boundaries, if not re-established after an injury by perfect healing, the epithelial cells once loose and free,

[1] Woodward on the Structure of Cancerous Tumors. Toner Lectures, Washington, 1873.

[2] Classen, Ueber Cancroid der Cornea, etc. Virchow's Archives, vol. 50, 1870.

there is nothing remaining to prevent them from travelling into surrounding connective tissue spaces and to thus form a cancer.

It is not the want of resistance of the surrounding tissue (as is generally held), but simply the getting loose of the normal cells from their place of attachment, which constitutes the formation of a malignant tumor.

It is the mobility of the cells, I think, that conditions the malignancy of a tumor. Any tumor, even the most benign lipoma, would be eminently malignant, if the cells composing it could get loose and travel through the widely open paths of the system of juice channels.

It would appear that I have deviated from the scope of my subject; but I think all the points considered have a direct bearing upon the etiology of tumors. Of course, I consider this communication nothing more than an attempt at the solution of the etiology of tumors. It is a complete compilation of all known facts and theories with a few additional original contributions. I hope it may suggest some thoughts and encourage others to undertake research on this subject, which, I believe, will establish the fact, that *all tumors are products of the inflammatory process, and that they should be considered as one of the terminations of inflammation.*[1] *April 26th*, 1881.

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5. *On swelling of the solitary and agminated lymphatic follicles of the small intestine in diseases other than enteric fever.*

Read by Dr. M. Longstreth, for Dr. Henry Dickson Bruns.[2]

During the week ending April 22d, 1880, three portions of small intestine exhibiting a swollen state of the lymphatic follicles, were brought into the Pathological Laboratory of the Pennsylvania Hospital.

One of these specimens was from a man, aged about 22 years, who died, as the temperature record, symptoms, and additional post-mortem evidence showed, of typhoid fever; another from a child, aged about three years, dying a few days after eating some ham of doubtful quality, from an acute catarrhal inflammation of the bowels; the third

[1] Owing to the length of this paper, its discussion was postponed until the following fall. This discussion, with an abstract of the paper, will appear in the next volume of the Society's Transactions.—Editor.

[2] See pp. 78 et seq. for details of two of these cases, with a discussion on the interpretation of the pathological conditions.

from a child, also aged about three years, who died of catarrhal pneumonia following an attack of measles.

These swollen glands suggested to Dr. Longstreth the following questions, to which he kindly called my attention, requesting me to furnish such answers to them as I might be able to make in the short time remaining before the next meeting of your Society.

Questions :—

(1.) Is a swollen state of the solitary and agminated lymphatic follicles of the small intestine ever met with in the bodies of those who have died of diseases other than enteric fever? And if so,

(2.) What diseases, other than enteric fever, give rise to this condition?

(3.) Is such a state of these glands more commonly seen in the bodies of children, and especially in the bodies of such as have died of disorders essentially infantile?

(4.) Does diarrhœa always coexist, as a symptom during life, with a swollen state of these glands?

(5.) Do the swollen follicles, in diseases other than enteric fever, ever run on into ulceration.

(6.) Is it possible microscopically or macroscopically, to distinguish these swollen glands (or the subsequent ulcerations) from the swollen glands of typhoid fever?

The first step was to look into the literature of the subject.

While numerous and elaborate descriptions of the condition of the ileum, and of its lymphatic follicles (solitary glands and Peyer's patches) in enteric fever, may be found in almost every work upon morbid anatomy and histology, here and there only are found scanty and unsatisfactory remarks upon the occurrence of a swollen condition of these glands in other diseases. I think, however, that to the first question we may answer :—

(1.) Yes, emphatically. Enteric fever is not the only disease in which a swollen condition of the glands of the small intestine and mesentery is found after death.

(2.) The diseases and morbid conditions, or at least some of them, which give rise to a swollen condition of these glands are :—

(*a.*) Acute and chronic catarrhal inflammation of the small intestine (Rokitansky,[1] Jones and Sieveking,[2] Wilks and Moxon,[3] Rind-

[1] Manual of Pathological Anat. Rokitansky, Sydenham ed., 1849, vol. ii. p. 88 *et seq.*

[2] Path. Anat. Jones and Sieveking, Payn. Am. ed., 1875, p. 587 *et seq.*

[3] Path. Anat. Wilks and Moxon. Sec. Am. ed., 1875, pp. 406, 635.

fleisch,[1] Orth,[2] Cornil and Ranvier[3]). (*b.*) Scarlet fever (Harley,[4] Bristowe,[5] Jones and Sieveking, Rokitansky). (*c.*) Measles (Cornil and Ranvier, Niemeyer,[6] Rokitansky state that this condition occurs in almost all the exanthemata. I can nowhere find special mention of it in measles). (*d.*) Smallpox (Rokitansky). (*e.*) Diphtheria (Dr. Wm. Squire,[7] Jones and Sieveking). (*f.*) Typhus fever (Dr. Geo. Buchanan,[8] Wilks and Moxon, Niemeyer). (*g.*) Hydrocephalic fever (Rokitansky). (*h.*) Erysipelas (Rokitansky). (*i.*) Puerperal fever (Cornil and Ranvier). (*j.*) Cholera (Rindfleisch, Dr. Edward Goodeve,[9] Orth, Jones and Sieveking, Rokitansky, Wilks and Moxon). (*k.*) Acute rheumatism. (*l.*) Gout. (*m.*) Croup. (*n.*) Suppurative and gangrenous diseases. (*o.*) Acute convulsions. (*p.*) Trismus and tetanus (Rokitansky).

(3.) As to whether this condition of the glands is more commonly found in the bodies of children or not, the data at my disposal does not enable me to say.

We must, however, remember that these glands are normally larger, more prominent, in children and young persons than in adults at and beyond the period of middle life, and are, in all probability, more active in function physiologically, and therefore more susceptible to pathological disturbance. Again, children and young persons are especially liable to most of the diseases cited in the above list, and in children that portion of the mucous membrane in which these glands are situated is wont, upon slight provocation, to take on catarrhal inflammation.

The only statements bearing upon this point, I find recorded by Hope,[10] who says: "I may refer to malignant cholera, in which I have

[1] Text-book of Path. Histology. Am. ed. Kloman and Miles, 1872, p. 336.

[2] Diagnosis in Path. Anat. Orth. Am. ed. Shattuck, Sabine, and Fitz, 1880.

[3] A Manual of Path. Histology. Cornil and Ranvier. Am. ed., 1880, p. 487 *et seq.*

[4] See art. Typhoid Fever, Reynolds's Syst. Med., Am. ed. Hartshorne, 1879.

[5] The Theory and Pract. Med. Bristowe. Am. ed. Hutchinson, pp. 161, 252.

[6] Text-book of Pract. Med. Niemeyer. Sec. Am. ed., 1870, vol. ii. p. 566.

[7] See art. Diphtheria. Reynolds's Syst. Med.

[8] See art. Typhus. Reynolds's Syst. Med.

[9] See art. Cholera. Reynolds's Syst. Med.

[10] Principles and Illustrations of Morbid Anatomy, by J. Hope, M.D., F.R.S London, 1834, p. 177.

almost invariably found the intumescence to affect children, while in adults it is of comparatively infrequent occurrence;" by Cornil and Ranvier, who writing of ileitis, state that in children the enlargement of Peyer's patches is very marked; and by Dr. Geo. Buchanan in the article on typhus in Reynolds's System of Medicine, where he remarks: "the agminated and solitary glands of both the large and the small intestines may be found enlarged, especially in children who have them naturally very visible."

Two of Dr. Longstreth's specimens were obtained from the bodies of children about three years of age.

(4.) The occurrence or non-occurrence of diarrhœa in cases in which we find enlargement of the solitary and agminated intestinal glands, *post mortem*.

Here the literature of the subject renders no assistance.

Further on, it will appear, I think it probable, that diarrhœa precedes or accompanies intumescence of the intestinal and mesenteric lymphatic glands.

Diarrhœa is a common symptom in nearly all the morbid conditions cited under (2).

In this connection it would be interesting to know if swelling of these glands is found after death in those cases of enteric fever, in which, instead of the more common diarrhœa we have a tendency to constipation.

(5.) As to ulceration of the intestinal glands in diseases other than enteric fever.

Jones and Sieveking record that in simple inflammation, chronic or acute, the enlargement of the intestinal glands "may go on to sloughing and ulceration."[1] And again, after mentioning that enlargement of the solitary and agminated glands may occur in cases of scarlet fever, they remark: "more rarely the process goes on to actual ulceration."[2] The same authors in treating of cholera,[3] say: "occasionally ulceration occurs, and small follicular ulcers are formed." MM. Cornil and Ranvier *a propos* of cholera write: "in a more advanced stage of the lesion when the autopsy is made during the period of revulsion, we sometimes find follicular ulcers situated at the location of the isolated follicles, or at the seat of Peyer's patches. These ulcerations may involve the deeper tissues to the extent of perforation (Hamernyck)."

[1] Loc. cit. p. 587. [2] Loc. cit. p. 599. [3] Loc. cit. p. 600.

On the other hand Dr. Geo. Buchanan contends that, "under no circumstances is there any deposit in these glands, nor does the ulceration of them, which is so common in typhoid, ever happen in typhus fever;" and Dr. Edward Goodeve asserts that "ulceration is not observed in simple cases" of cholera.

(6.) Is it possible to distinguish by macroscopic or microscopic means the swollen intestinal glands of cholera, scarlet fever, etc., from those of enteric fever.

The specific character of the lesion in typhoid fever has so long been regarded as an established pathological fact, that it is with the utmost hesitation I venture to record to the above question an answer in the negative. Nevertheless I must do so, founding my belief, however, not upon any microscopical attainments of my own, which are indeed nil, but upon the authority of others entitled to be heard, and upon a rational consideration of the prime facts in the case.

Dr. John Harley, speaking of the swollen glands of scarlet fever, says: "after most careful microscopical examination of these swollen glands, I have failed to distinguish the slightest difference between them and those of the first period of enteric fever."[1]

Dr. Longstreth assures me that he has repeatedly examined swollen intestinal and mesenteric glands in various diseases, and that he has never been able to perceive any essential difference between them and those of enteric fever. Almost all the authorities cited above as mentioning the occurrence of swelling of these glands in various diseases, accord them no further consideration, while they give most detailed accounts of their macro- and microscopical appearance in typhoid, and occasionally also in cholera, appearing to regard the entirely different nature of the two conditions as a fact established beyond the peradventure of a doubt.

The German authors even go so far as to describe a specific typhoid cell. Here we must remember that a man, be he never so honest, examining a morbid condition concerning the specific nature of which he has strong preconceived opinions, would in all probability discover in his specimen a specific element or arrangement.

Of our own specimens I was not so fortunate as to see, before section, the one from the case of catarrhal inflammation of the intestine, but I am informed by Dr. Longstreth that it presented appearances entirely similar to the specimen from the case of measles.

[1] Loc. cit. p. 223.

The latter was a single Peyer's patch much swollen, about three inches long, one and a half broad, and two lines thick, presenting in every particular the appearance of the oft-described *plaque réticulée.* A section showed the gland densely packed with lymphoid-cells; the villi and tubular glands (Lieberkühn's), which covered the enlarged follicle almost to the apex, were hypertrophied. Scattered here and there throughout the villi were round cells which had taken the coloring matter (all the sections were stained with carmine) more deeply than the surrounding cells. In a section obtained from the specimen of catarrhal inflammation, the mucous membrane was almost entirely denuded of villi and tubular glands. Otherwise the microscopic appearances were entirely similar.

I believe the pathology of all these glandular swellings to be simply and briefly this:—the solitary and agminated glands of the intestine are lymphatic glands[1]—their function is absorbative, not secretive.

It is well known that where an area from which the lymph stream is conveyed into certain lymphatic glands, suffers inflammatory or other irritation, these glands become swollen, enlarged.

Now nearly all the diseases enumerated under (2) are due to blood poisoning, and have as a common symptom (whether the result of an abnormal condition of the blood, a capillary dilatation dependent upon nervous influence, or an effort to eliminate the poison concerns us not) diarrhœa and congestion or inflammation of the intestinal mucous membrane. As the result of this the cells of the lymph follicles imbedded in the membrane proliferate—the glands become swollen.

It is probable that the glands are found more markedly and frequently swollen in enteric fever than in other diseases, because here the cause has longer to act. We have here a chronic inflammation, so to speak, a slow, continuous, irritation of the mucous membrane.

Here also the toxic agent acts, probably, directly upon the glands, is absorbed in the intestine, which is not the case, we believe, in the other diseases.

For the same reasons, I believe, ulceration is more common in enteric fever. The inflammation is of a lower grade, is slower. Acute inflammations are more apt to end in resolution than chronic ones. The general and with it the lymphatic circulation, enfeebled by the long typhoid illness, is inadequate to the task of bearing away the

[1] Stricker's History—Am. Trans. 1872, p. 383.

multitudinous cells of the tumid follicles. Hence, by reason of continued pressure upon vessels, etc., we have sloughing of the gland.

A very high grade of acute inflammation may act in like manner.

There are too many cells in proportion to the amount of nutritive material which can be provided.

Thus we have ulceration occurring in cholera. Note, however, that it is not found save in the bodies of those who have died at a late stage. Recall here also, what has already been said in regard to more severe irritation by direct action of the poison in typhoid. I have already called attention to the fact that these glands are, in all probability, functionally more active than in adults. In this connection the remark of Dr. Frierick, of Dresden,[1] that ulceration of these glands in cases of typhoid occurring in children is extremely rare, possesses a high degree of interest.

MM. Cornil and Ranvier, who do not hold the idea of a specific typhoid cell, describe with great care the appearances of the intestinal mucous membrane and glands in typhoid and cholera. In the first, besides the engorgement of the follicle with cells, the villi and tubular glands are hypertrophied and distorted.

Now this is precisely the condition existing in the section from the case of measles. In cholera, say our authorities, the mucous membrane is found stripped of its epithelium, villi, and crypts.

This condition was found in the specimen from the case of catarrhal inflammation.

These two states are by no means specific, but depend, I think, upon two varying factors; the lapse of time between death and the cadaveric section, and the severity of the diarrhœa previously existing as a symptom.

When we examine a body but a short time after death, we expect to find the epithelium, villi, and tubular glands in a much better state of preservation than if we had allowed a longer time to elapse. Again, if the grade of intestinal inflammation has been high, the diarrhœa severe, sweeping, the epithelium loosened by rapid proliferation, who would not expect to find the mucous membrane denuded?

Now all these conditions are present in cholera, but they were also present in the specimen illustrating a high degree of catarrhal inflammation, and this specimen was obtained some time after death, hence the similarity. In the opposite condition, where the inflamma-

[1] Aitken's Pract. Med., third Am. ed., 1872, p. 105, *et seq.*

tion is of lower grade, of longer duration, and the diarrhœa not nearly so severe, we find, as in the typhoid state described by Cornil and Ranvier, and in our specimen from the case of measles (taken sooner after the death of the patient than the other) the mucous membrane thickened and its appendages hypertrophied.

In conclusion I would call attention to the importance of this question. A glance at a few of the works upon Morbid Anatomy and Histology and the Practice of Medicine, will show how much weight is laid upon this lesion in connection with enteric fever, and how lightly it is passed over in the case of other morbid conditions. Thus, the student comes to regard the lesion as pathognomonic, and it seems very probable that many a *post-mortem* diagnosis of typhoid fever is made to depend solely upon its presence.[1] *May 12th*, 1881.

[1] See (loc. cit.) the cases given by Dr. John Harley, under the heading Associated Pathology of Typhoid Fever.

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